The optic nerve and the accessory sinuses of the nose / By. Professor A. Onodi. Authorized translation by J. Luckhoff. With 50 illustrations.

THE OPTIC NERVE
AND
THE ACCESSORY SINUSES OF THE NOSE

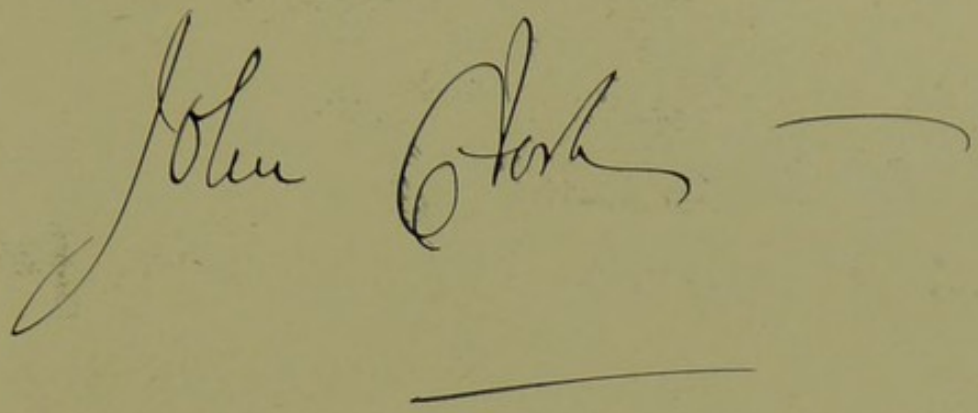
A. Onodi—J. Lückhoff

logos = science or knowledge

topography = topos = place
graphein =

spheroidal = sphera - oidos = form

traumatic = trauma = injury -



THE OPTIC NERVE

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THE ACCESSORY SINUSES OF THE NOSE

A CONTRIBUTION TO

THE STUDY OF CANALICULAR NEURITIS AND ATROPHY OF THE OPTIC NERVE OF NASAL ORIGIN

BY

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AUTHORIZED TRANSLATION

BY

J. LÜCKHOFF, M.D. EDIN., CH.B.

CAPE TOWN

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1910

PREFACE TO THE ENGLISH EDITION

THE German edition of my book was published in 1906, and contained twenty-seven illustrations only. While personally discussing the English translation with my honoured colleague, Dr. Lückhoff of Cape Town, I decided to make considerable additions to that number, thus rendering the work more complete and instructive. Instead, therefore, of only twenty-seven illustrations, the English edition contains fifty. In order to bring it quite up to date, I have also made use of the addresses which I delivered on the subject at the First International Laryngological Congress held in Vienna in 1908, and at the Annual Meeting of the Academy of Ophthalmology, Rhinology, and Oto-laryngology in New York in 1909. It is my pleasant duty on this occasion to express my gratitude to Dr. Lückhoff for the care he has devoted to the translation, to Dr. Lamb of Birmingham for his kind assistance in seeing the proofs through the press, and to the publishers for the handsome appearance they have given to my book.

A. ÓNODI.

BUDAPEST,
February, 1910.

PREFACE TO THE GERMAN EDITION

THE minute anatomy of the nasal accessory sinuses and their relations to the optic nerves have now engaged my attention for a period of ten years. The results I have obtained provide an anatomical basis for the study of visual disturbances and blindness, associated with disease of the accessory sinuses, and especially with disease of the posterior ethmoidal cells and the sphenoidal sinuses. An accurate study of the pathological anatomy of this subject is still required. To this and to accurate clinical observation we must look for the filling up of the numerous gaps in our knowledge of this subject. To my honoured colleagues I pass on the result of these researches, in the hope that it may stimulate others to pursue this important subject to a successful issue, both through ophthalmic and rhinological research.

My illustrations have been reproduced from photographs, natural size. I demonstrated ten of the specimens at the Thirty-third Ophthalmological Congress, held at Heidelberg in 1906. Eight specimens reproduced in my atlas, "The Accessory Sinuses of the Nose," I have again utilized in this treatise.

A. ÓNODI.

BUDAPEST.

THE OPTIC NERVE

AND THE

ACCESSORY SINUSES OF THE NOSE

I

TOPOGRAPHICAL ANATOMY

Up to the present, the more intimate relationships of the optic nerves to the nasal accessory sinuses, and their topographical anatomy, have not been thoroughly worked out. In textbooks on anatomy, in the description of the course of the optic nerves in the base of the skull, their association with the accessory sinuses is naturally pointed out.

In their monograph on the relationship of the optic nerves to the sphenoidal sinus, Berger and Tyrman[1] give three illustrations which demonstrate the variation in the thickness of the sphenoidal sinus walls at the level of the optic foramen. Further, they show two skulls "in which the ethmoidal labyrinth expanded itself posteriorly to such a degree that the anterior sphenoidal wall lay in a position posterior to the optic canals, so that these no longer bordered on the sphenoidal sinus." Zuckerkandl[2] and Douglas[3] have described the sphenoidal sinuses in their relation to the lesser wings of the sphenoid. Such, covering a long period of years, are the antecedent investigations.

I have already in my published works demonstrated the frequent and intimate relationship of the optic nerves to the most posterior ethmoidal cell, and have for this reason pointed out that we must relinquish the rigid acceptation of the relationship of the optic nerves to the sphenoidal sinus.

The investigations completed by me up to the present have established thirty-eight variations in the relationships of the optic nerves to the most posterior ethmoidal cell, and the sphenoidal sinus. When

[1] "Diseases of the Sphenoid and of the Ethmoid Labyrinth," 1886.
[2] "Anatomy of the Nasal Cavities," 1882, 1893.
[3] *Monatsschrift für Ohren- und Kehlkopfkrankheiten*, 1897.

we consider the embryological relationships of the optic sulcus and optic canal, and pay attention to the varieties of form which individual parts of the sphenoid, such as the præsphenoid, orbito-sphenoid, and

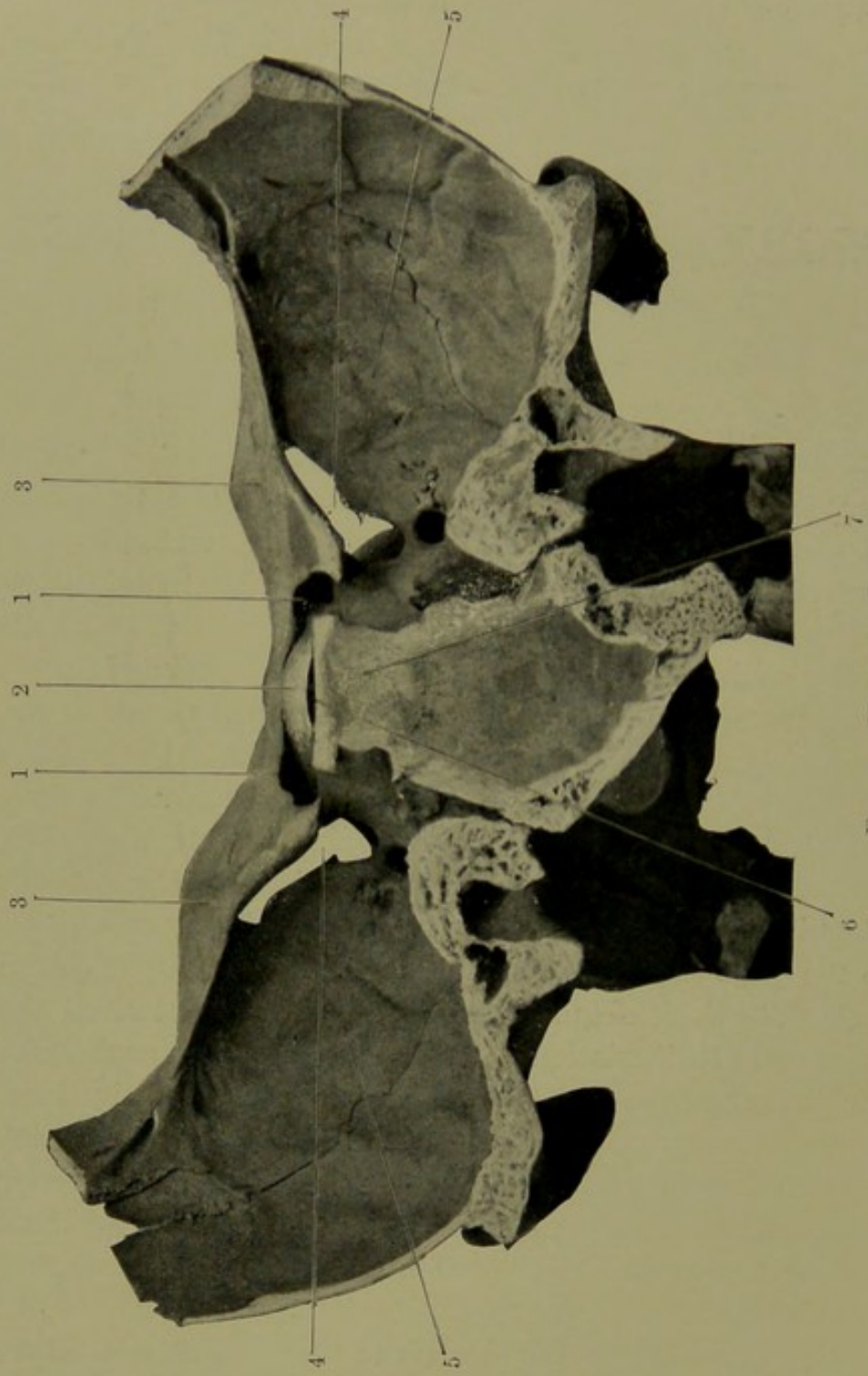


FIG. 1.—NATURAL SIZE.

1. Optic foramen.
2. Optic sulcus.
3. Small wing of sphenoid.
4. Sphenoidal fissure.
5. Large wing of sphenoid.
6. Sella turcica.
7. Dorsum sellæ.

basi-sphenoid, show both in fœtal and post-natal periods, then such variations become intelligible. And when we more carefully consider the development of the sphenoidal sinuses, the appearance of the muscles attached to the sphenoid, and the development of the

ethmoidal cells and frontal sinus, as well as the appearance of the ossification centres, which already in fœtal and post-natal periods produce variations in shape and relationship, then we can explain the large number of varieties, and the striking peculiarities which I have found, and which I will discuss later. At present I do not wish

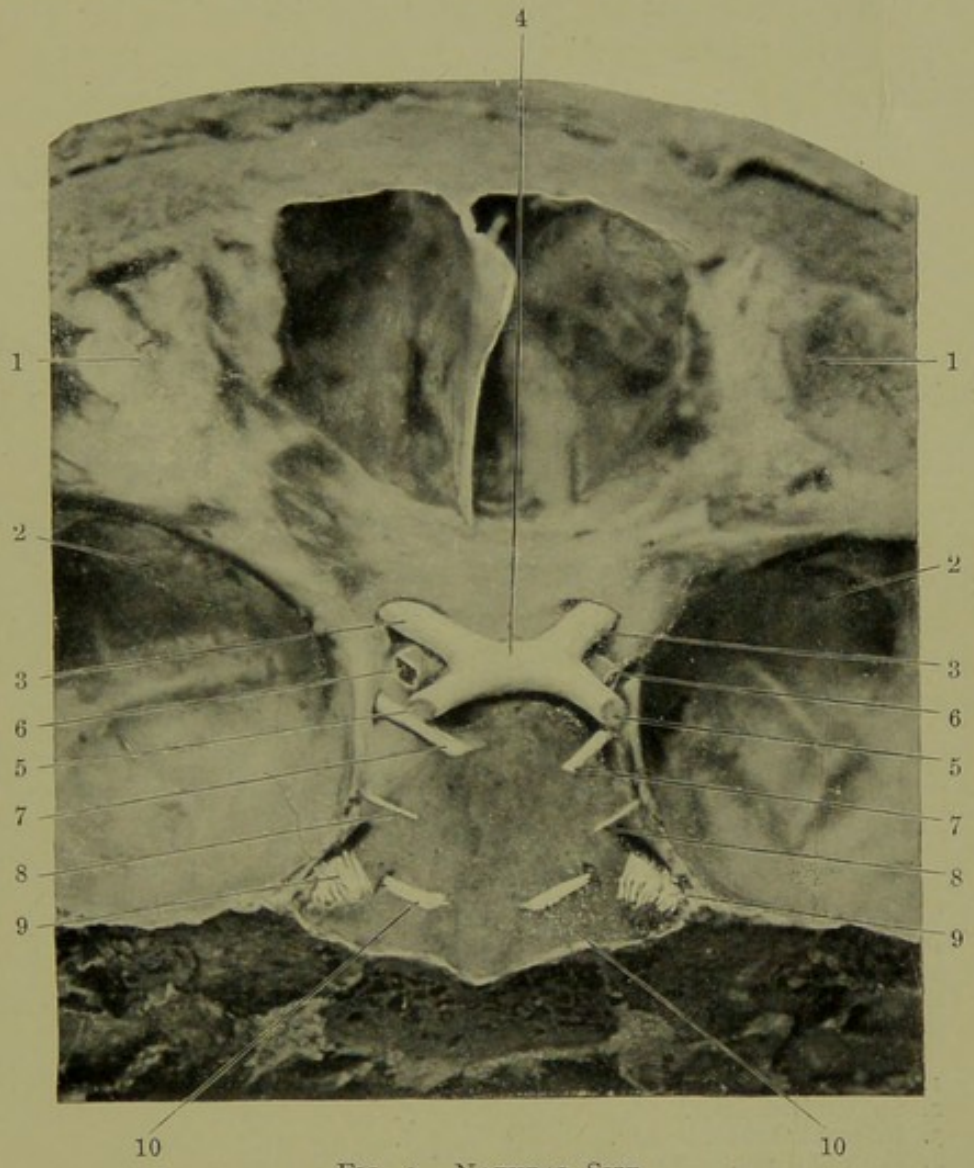


FIG. 2.—NATURAL SIZE.

1. Roof of orbit.
2. Middle cranial fossa.
3. Optic tract.
4. Optic chiasma.
5. Optic nerve.
6. Internal carotid artery.
7. Third nerve—oculo-motor.
8. Fourth nerve—trochlearis.
9. Fifth nerve—trigeminus.
10. Sixth nerve—abducens.

to enter upon a close analysis of the morphological relationships. It will be sufficient to define the topographical relationships of the optic nerves, optic canal, and optic sulcus, to the posterior ethmoidal cells and the sphenoidal sinuses.

The thirty-eight different varieties which I have found will fall

into twelve main groups. These I will describe individually, with the aid of photographs of the specimens, which are reproduced in natural size. In order, later, to have a clearer view of the varying relationships of the optic nerves, optic sulcus, optic foramina, and optic canals, to the posterior ethmoidal cells and sphenoidal sinuses, I will proceed first to illustrate the position of these in the base of the skull.

Figure 1 illustrates on a bone preparation the region of the optic foramina and optic sulci. The small wing of the sphenoid or ala

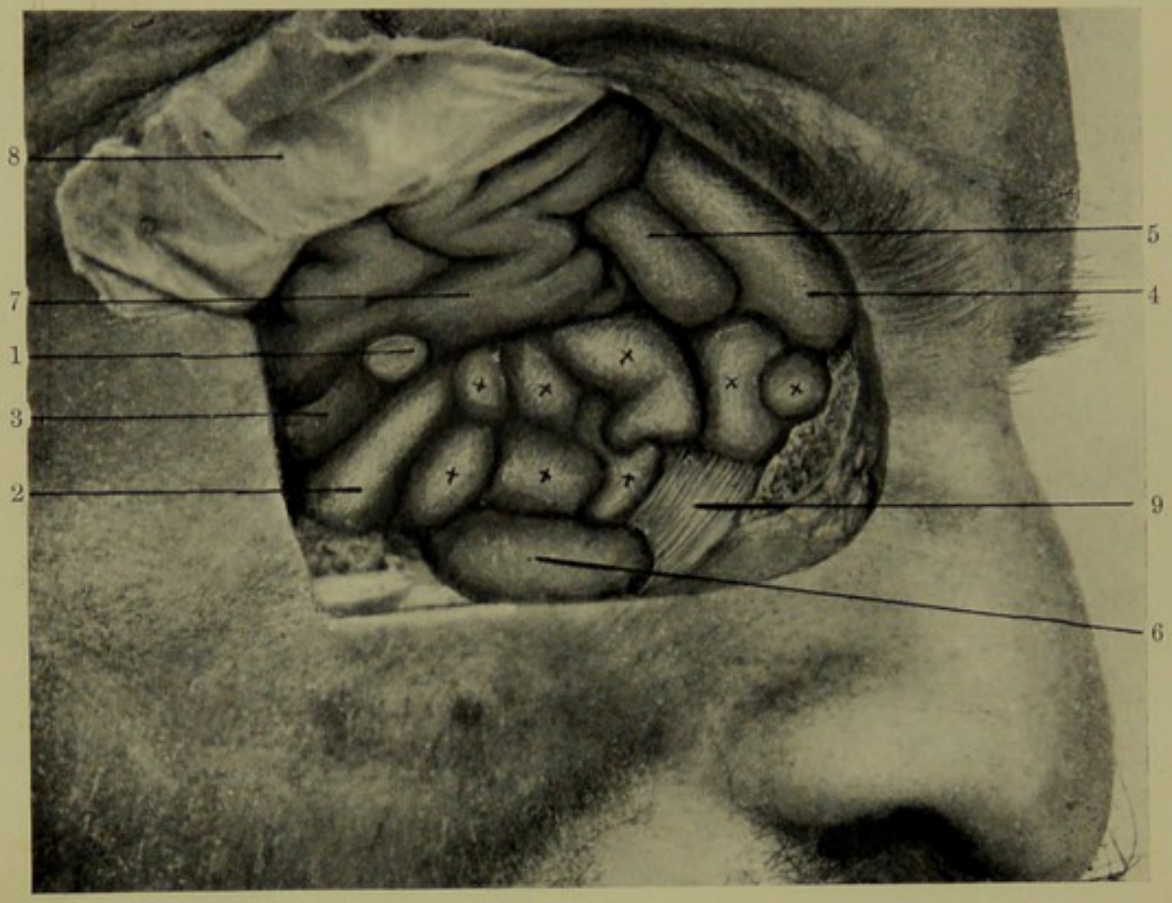


FIG. 3.—NATURAL SIZE.

1. Optic nerve.
2. Sphenoidal sinus.
3. Internal carotid artery.
4. Frontal sinus.
5. Orbital recess of frontal sinus.
6. Maxillary antrum.
7. Frontal lobe.
8. Dura mater.
9. Nasal cavity.

× × × × × × × ×, Ethmoidal cells.

parva bridges the sphenoidal fissure on each side, and medial to this is the optic foramen, which prolongs itself into a short optic canal. The roofs of the optic foramina together form a low ridge, which is called the "limbus sphenoidalis," and forms the boundary of the jugum sphenoidale. The optic sulcus lies between these and the sella turcica.

Figure 2 illustrates the optic nerve as it enters the optic foramen of its side, and also the chiasma and optic tract. The flattened bands

which form the optic tracts unite and cross in the chiasma, which lies in the sulcus, between the sphenoidal limbus and the tuberculum sellæ. From the chiasma pass the optic nerves, to extend into the

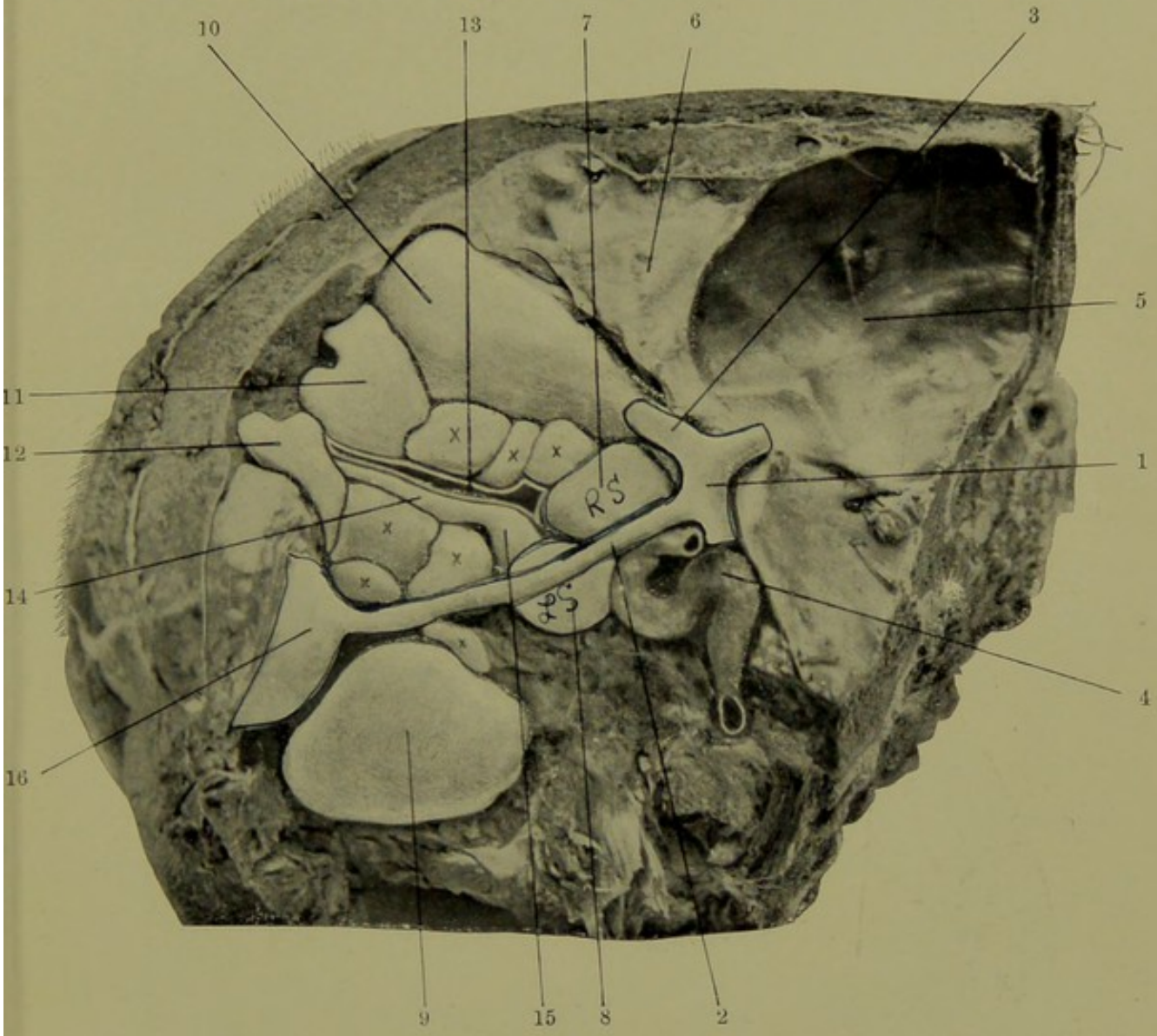


FIG. 4.—NATURAL SIZE.

1. Optic chiasma.
2. Left optic nerve.
3. Right optic nerve.
4. Internal carotid artery.
5. Middle cranial fossa.
6. Anterior cranial fossa.
7. Right sphenoidal sinus.
8. Left sphenoidal sinus.
9. Left maxillary antrum.
10. Periosteum of orbit.
11. Right frontal sinus.
12. Left frontal sinus.
13. Nasal cavity.
14. Olfactory fissure.
15. Spheno-ethmoidal recess—left.
16. Eyeball.

× × × × × ×, Ethmoidal cells.

optic foramina, above the ophthalmic arteries. The illustration further shows the internal carotid in its course to the brain, as well as the trunks of the oculo-motor, trochlear, trigeminal, and abducens nerves.

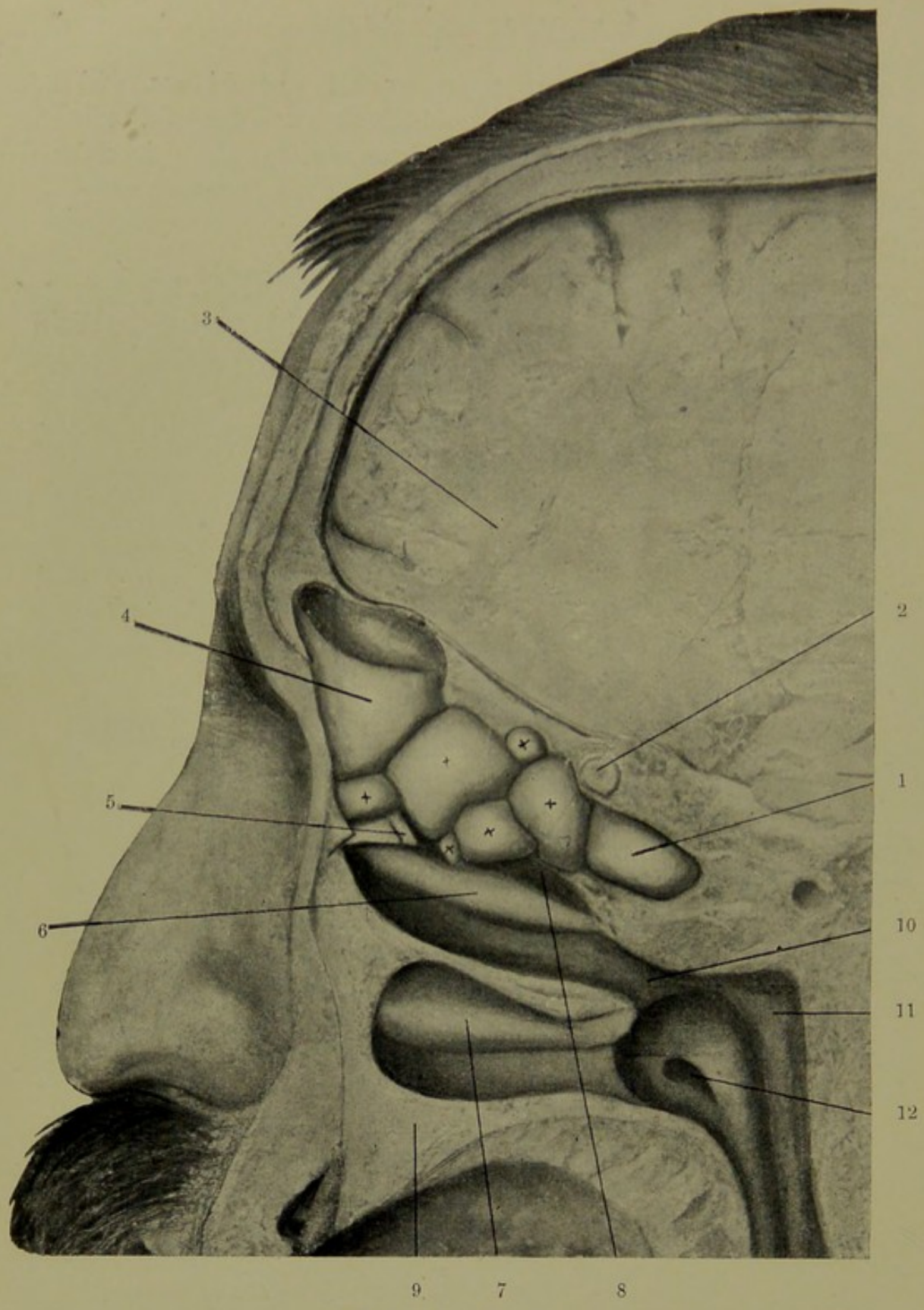


FIG. 5.—NATURAL SIZE.

1. Sphenoidal sinus.
2. Optic nerve.
3. Frontal lobe.
4. Frontal sinus.
5. Unciform process.
6. Middle turbinal.
7. Inferior turbinal.
8. Superior meatus.
9. Palato.
10. Septum.
11. Naso-pharyngeal cavity.
12. Eustachian tube.

× × × × ×, Ethmoidal cells.

In Figures 2 and 104 (Atlas)[1] the optic tract is shown *in situ* in its relationship to the base of the brain. Figure 2, in sagittal section, illustrates the position of the optic tract in its relationship to the sphenoidal sinus. Figure 104 illustrates, in sagittal section, a specimen which had been injected with a 10 per cent. formalin solution, in order to retain the mucous membrane of the individual sinuses in its saccular form. In it are seen *in situ* the maxillary, frontal, ethmoidal, and sphenoidal sinuses, as well as the optic tract in its relationship with the base of the brain and with the sphenoidal sinuses.

Figure 3 illustrates a formalin-hardened specimen with the structures that lie in touch with the walls of the orbital cavities exposed to view. The bone of these walls having been removed, the mucous membrane of the accessory sinuses is shown in its natural shape, as well as the under surface of the frontal lobes of the brain, and the optic nerve and internal carotid artery. The position of the optic nerve and its intimate relation to the sphenoidal sinus is seen.

Figure 4 illustrates a formalin-hardened specimen. In it can be seen the position of the optic chiasma, and the course of the left optic nerve up to its entry into the eyeball. This nerve touches upon the left sphenoidal sinus. The different accessory sinuses, in saccular form, are also shown. On the left side, in addition, appears the roof of the left nasal cavity, and on the right the sinuses in their relation to the periosteum of the orbit.

Figure 5 illustrates a specimen hardened in formalin, in which the nasal cavities have been opened from without. It shows in sagittal section the accessory sinuses, and the optic nerve in its association with the posterior ethmoidal cells and sphenoidal sinuses.

In those cases in which the wall of the optic canal is formed out of the wall of the most posterior ethmoidal cell, the partition between the canal and sinus is usually thin, and may in some cases be very thin. In one case it measured 1 millimetre, in another 2 millimetres, in thickness. In those cases in which the walls of the optic sulcus and optic canals are formed by the wall of the sphenoidal sinuses the variation in thickness may range between 1, 2, 3, 4, 5, 9, and 12 millimetres. Later I will discuss more in detail the practical importance of these facts, and of dehiscences which may be present in the optic canal, as well as the importance which may attach to an optic canal,

[1] Ónodi. "Accessory Sinuses of the Nose," by Hölder, Vienna.

the course of which lies within the sphenoidal sinus and posterior ethmoidal cell. The length of the optic canal may reach 6, 8, 10, or 12 millimetres.

Group 1.

1. To this group belong those cases in which neither the most posterior ethmoidal cell nor the sphenoidal sinus stands in close relationship with the optic canal or optic sulcus.

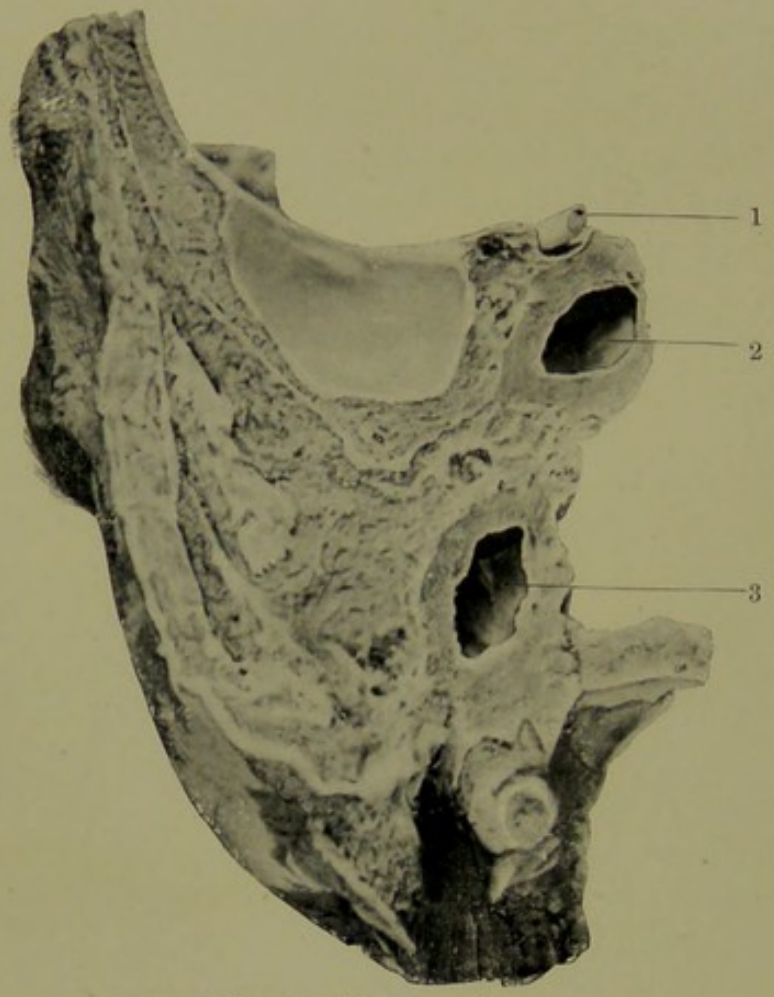


FIG. 6.—NATURAL SIZE.

1. Optic nerve. 2. Sphenoidal sinus.
3. Maxillary antrum.

Figure 77 (Atlas) illustrates a specimen in frontal section, in which the right sphenoidal sinus is 12 millimetres long, 10 millimetres broad, and 9 millimetres high, and the left is 9 millimetres long, 10 millimetres broad, and 20 millimetres high. The ostium of each sinus is 1 millimetre in diameter, and the layer of bone intervening between the sphenoidal sinuses and the optic nerves and chiasma in the optic canals and sulcus measures from 8 to 12 millimetres in thickness.

Figure 6 likewise shows a specimen in frontal section, in which bone 6 millimetres in thickness separates the optic nerve from the sphenoidal sinus and the ethmoidal cell. On the surface of the section are to be seen the optic nerve, sphenoidal sinus, and maxillary antrum.

Figure 76 (Atlas) illustrates a frontal section at the level of the posterior border of the nasal septum, in which bone 8 millimetres in thickness separates the sphenoidal sinus from the hypophysis. Above this lie the optic tracts and optic chiasma.

Group 2.

There are three varieties of relationship between the ethmoidal cells and the optic nerves, which I class under this group.

2. The inner and lower walls of the optic canal may be formed out of the wall of the most posterior ethmoidal cell.

Figure 7 illustrates this relationship. Here the sphenoidal sinuses are placed beneath the posterior ethmoidal cells, and stand in no relation to the optic nerves. The ostia of the sphenoidal sinuses open below the level of the floor of the posterior ethmoidal cells.

On the other hand, the inner and lower walls of the optic canal border on the ethmoidal cells, this relation being maintained up to the level of the optic foramina.

The width of the thin partition between the sphenoidal sinuses and ethmoidal cells is 20 millimetres on the right side, and 15 millimetres on the left side. The anterior walls of the sphenoidal sinuses are extremely small.

3. The inner wall of the right optic canal is formed by the wall of the right posterior ethmoidal cell, and the inner and lower walls of the left optic canal, and also the left third of the optic sulcus, are formed by the wall of the left posterior ethmoidal cell.

4. The inner and lower walls of the right optic canal and the wall of the middle and right third of the optic sulcus are formed by the wall of a large ethmoidal cell on the right side. The inner wall of the left optic canal and the wall of the left third of the optic sulcus are formed by the wall of a large ethmoidal cell on the left side, which unites with the frontal sinus of that side.

Figure 8 illustrates such a specimen in horizontal section.

This is a structural and not a pathological anomaly, for the structures everywhere show normal relations, and the openings of the sinuses are symmetrical on the two sides.

On the left side the ethmoidal cells have not only coalesced into one cavity, but this cavity is also continuous with the left frontal sinus. In this manner is formed a cavity 62 millimetres in length. The frontal sinus itself is 45 millimetres broad and 65 millimetres in height. Its anterior wall is 7 millimetres in thickness.

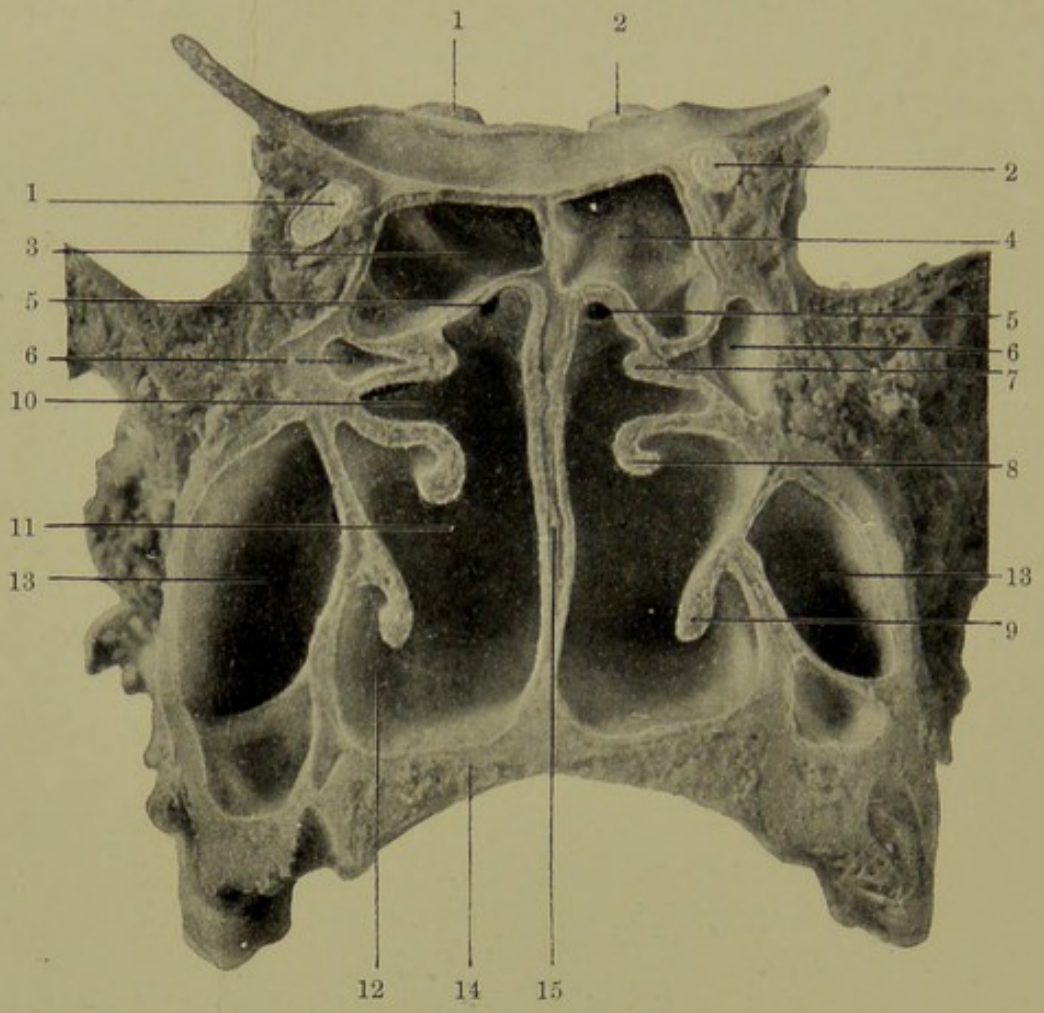


FIG. 7.—NATURAL SIZE.

1. Right optic nerve.
2. Left optic nerve.
3. Posterior ethmoidal cell—right.
4. Posterior ethmoidal cell—left.
5. Ostium of sphenoid.
6. Ethmoidal cell.
7. Superior turbinal.
8. Middle turbinal.
9. Inferior turbinal.
10. Superior meatus.
11. Middle meatus.
12. Inferior meatus.
13. Maxillary antrum.
14. Hard palate.
15. Septum.

On the right side the ethmoidal cells coalesce to form a cavity 45 millimetres long. It is separated from the right frontal sinus. It contains two recesses. The greatest breadth of the right and left ethmoidal cavities is 25 millimetres.

The right frontal sinus is 23 millimetres long, 40 millimetres broad, and 40 millimetres in height. This cavity has a recess. Its anterior wall is $1\frac{1}{2}$ millimetres thick.

On the right side a sphenoidal sinus is in evidence.

Distant 23 millimetres from the anterior wall of the frontal sinus, on each side, is the opening of the anterior ethmoidal foramen. This is continued downwards in the lateral wall of the frontal sinus as a groove 16 millimetres in length, called by me the "semicanalis ethmoidalis." On each side, above the anterior part of the hiatus semilunaris, the frontal sinus opens with an aperture 9 millimetres long and 4 millimetres broad.

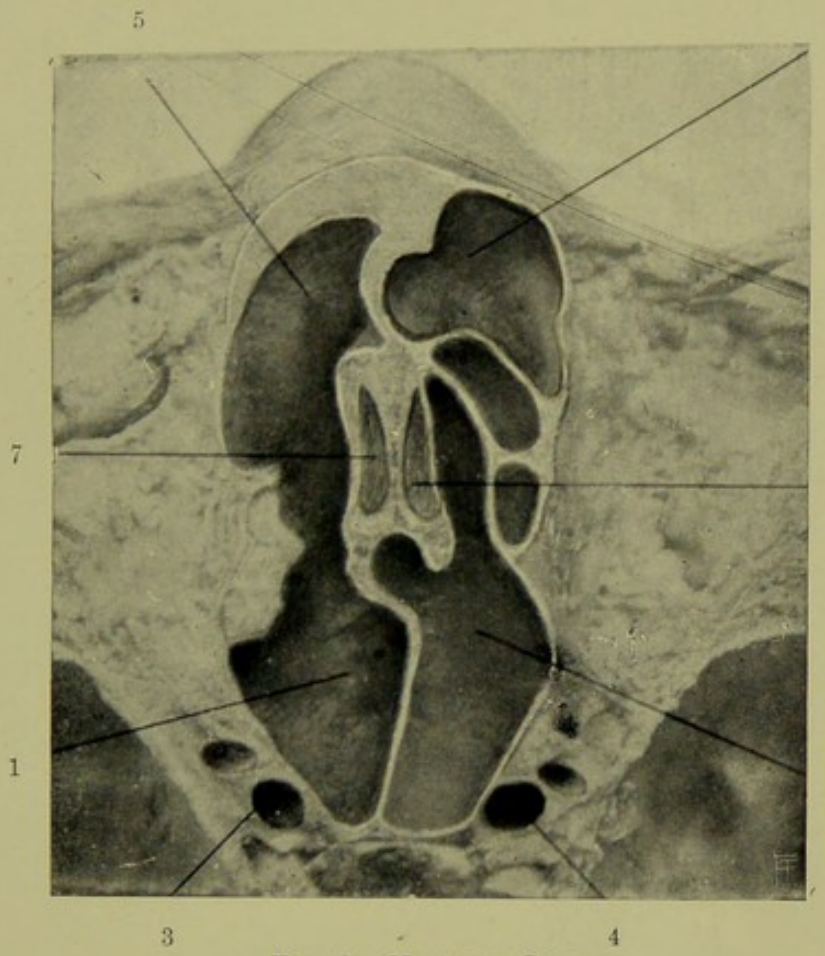


FIG. 8.—NATURAL SIZE.

1. Left ethmoidal cavity.
2. Right ethmoidal cavity.
3. Left internal carotid.
4. Right internal carotid.
5. Left frontal sinus.
6. Right frontal sinus.
7. Left olfactory fissure.
8. Right olfactory fissure.

Into the posterior end of the hiatus semilunaris the maxillary antrum opens. Both the unciform process and the ethmoidal bulla are well defined.

The superior turbinate is formed by a ridge passing from above the middle turbinate bone, and in the groove between these the ethmoidal cells open. This ostium is placed symmetrically on the two sides in the superior meatus.

Group 3.

In this group the most posterior ethmoidal cell is in relation to the optic nerve. There are seven varieties to be distinguished. In all of these the cell is in relation to the nerve of the same side, but in two it is in relation also with the optic nerve of the opposite side.

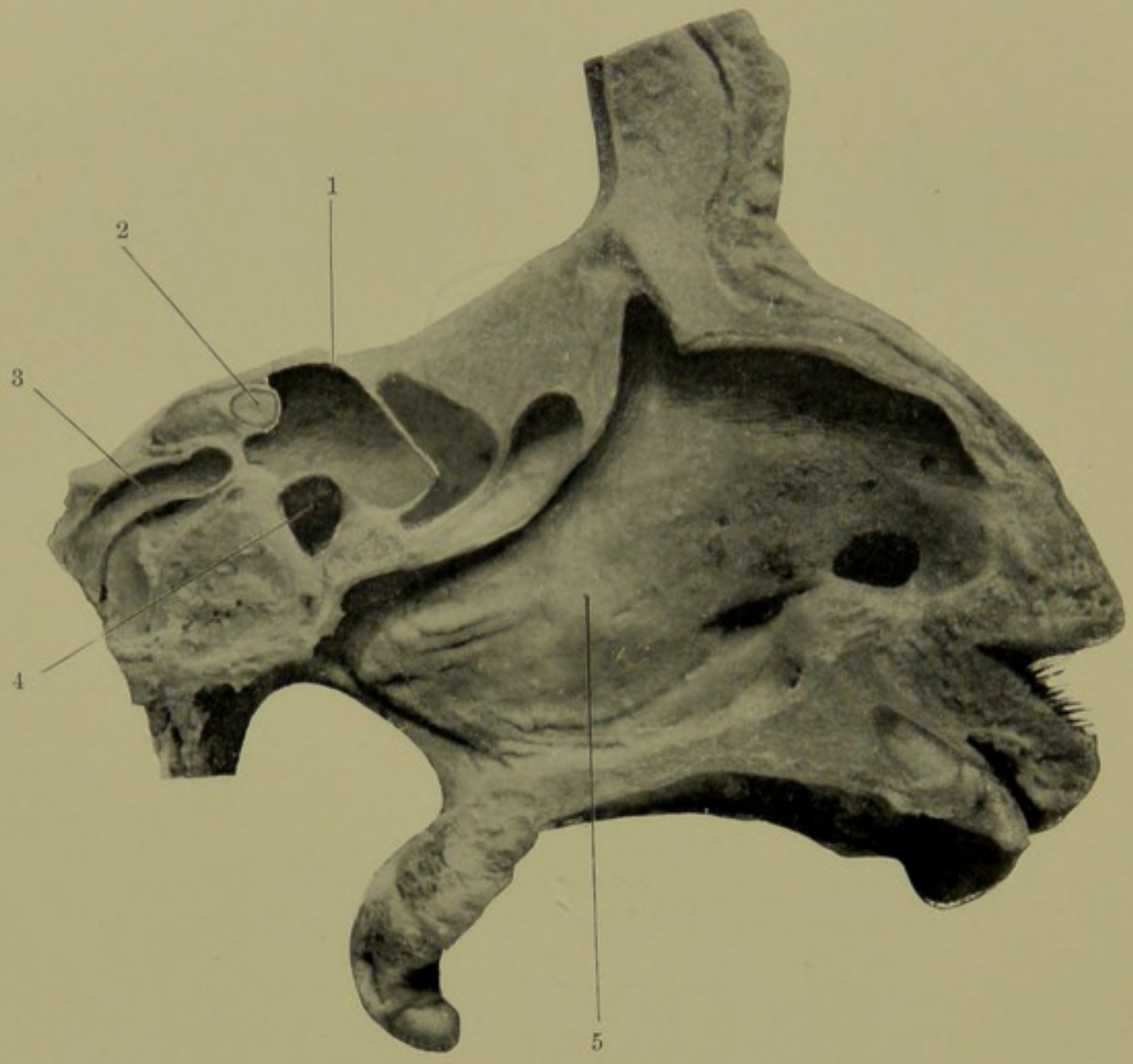


FIG. 9.—NATURAL SIZE.

1. Posterior ethmoidal cell.
2. Optic nerve.
3. Internal carotid artery.
4. Sphenoidal sinus.
5. Septum.

5. The inner, lower, and outer walls of the right optic canal, and the right third of the optic sulcus, are formed by the wall of the posterior ethmoidal cell of that side.

Figure 17 (Atlas) shows this arrangement, in sagittal section. The large sphenoidal sinus is here in no relation to the optic sulcus,

canal, or foramen. It is 22 millimetres in length and 18 millimetres high. The most posterior ethmoidal cell is 30 millimetres long, and from the middle line 18 millimetres in breadth. This cavity passes under the optic sulcus and the optic foramen into the lesser wing of the sphenoid. It forms the whole wall of the sulcus and the inner, lower, and outer walls of the optic canal of that side.

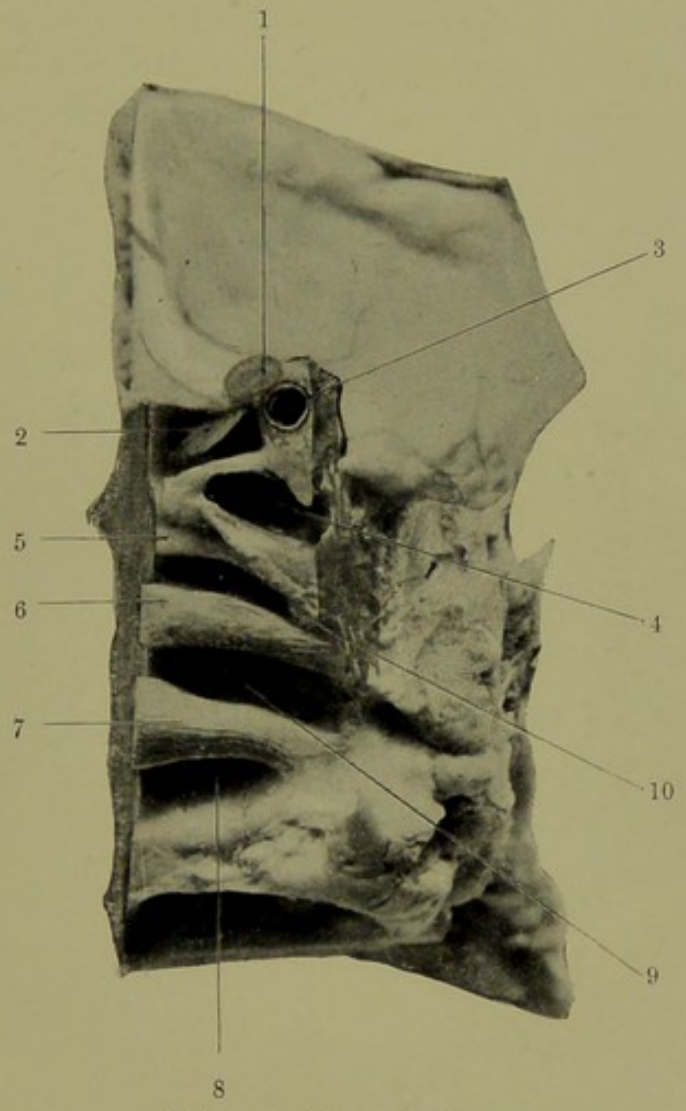


FIG. 10.—NATURAL SIZE.

1. Optic nerve.
2. Posterior ethmoidal cell.
3. Internal carotid artery.
4. Sphenoidal sinus.
5. Optic canal.
6. Superior turbinal.
7. Middle turbinal.
8. Inferior meatus.
9. Middle meatus.
10. Superior meatus.

6. The inner and lower walls of the left optic canal and the whole of the optic sulcus are formed by the most posterior ethmoidal cell of that side.

Figure 16 (Atlas) illustrates this on a bone specimen, in sagittal section. The posterior ethmoidal cell is 11 millimetres long, 14 milli-

metres broad from the middle line, and 13 millimetres high. The relation of the optic canal and sulcus is as above. The sphenoidal sinus, though large, is not associated with the optic canal or optic sulcus. Its measurements are 25 millimetres long, 15 millimetres high, and from the middle line 15 millimetres broad.

7. The inner and lower walls of the optic canal are formed by the wall of the most posterior ethmoidal cell of the same side.

Figure 13 (Atlas) demonstrates, in sagittal section, a posterior ethmoidal cell on the left side, which for 6 millimetres stretches back

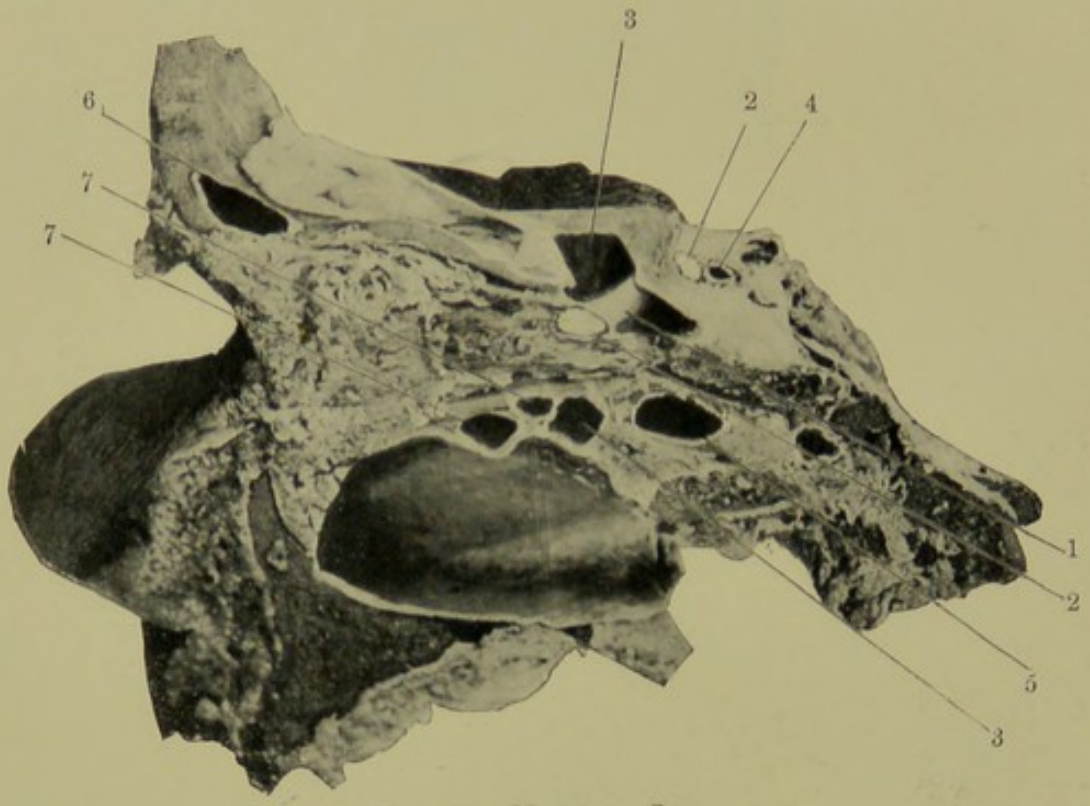


FIG. 11.—NATURAL SIZE.

1. Optic canal.
2. Optic nerve.
3. Posterior ethmoidal cell.
4. Internal carotid artery.
5. Sphenoidal sinus.
6. Frontal sinus.
7. Ethmoidal cells.

underneath the optic canal. The length of this cell is 34 millimetres, and its height is 16 millimetres. The relations of the optic nerve are shown. The accessory sinuses and the nasal cavity have in this specimen been exposed to view from without.

8. The upper, inner, and lower walls of the optic canal are formed by the most posterior ethmoidal cell.

Figures 9 and 10 demonstrate this relationship. In Figure 9, in sagittal section, it is seen that the optic canal is surrounded on the inner side from roof to floor by the posterior ethmoidal cell. In

Figure 10 is represented a specimen, in frontal section, in which the accessory sinuses have been opened from the front and from the nasal cavity. On the right side is seen the optic canal, lying for 12 millimetres of its course in the outer wall of the right posterior ethmoidal cell. The cell extends from above the canal round to its inner and lower side. The optic nerve on the right lies beside the internal carotid artery, and is shown in its course up to the optic foramen.

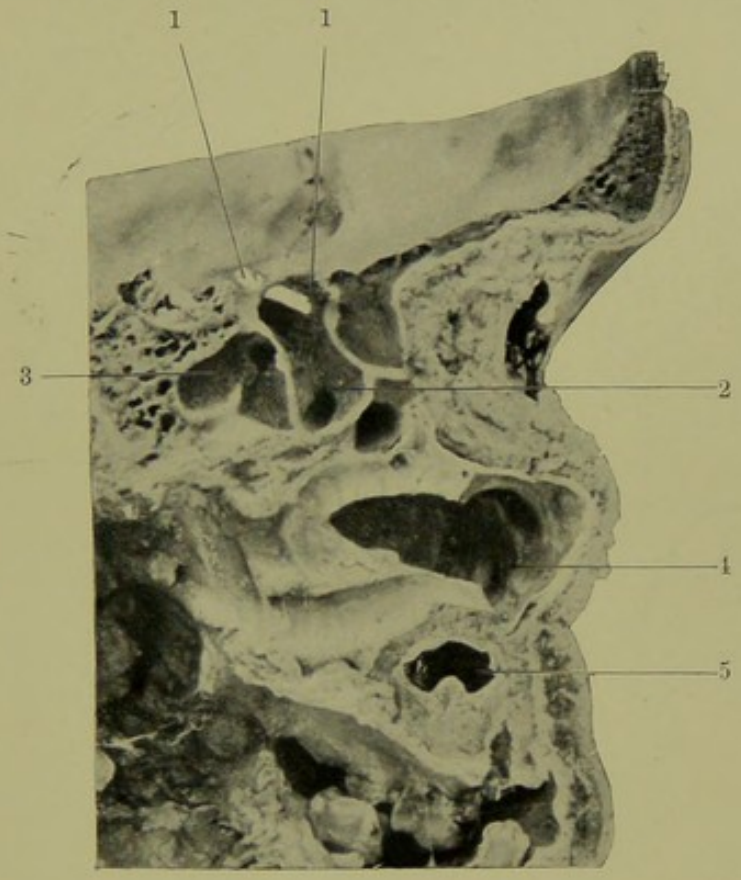


FIG. 12.—NATURAL SIZE.

1. Optic nerve.
2. Posterior ethmoidal cell.
3. Sphenoidal sinus.
4. Maxillary antrum.
5. Alveolar cyst.

Figure 11 demonstrates, in sagittal section, an optic canal on the right side, of 10 millimetres length, which has its course in a right posterior ethmoidal cell. This cell is 15 millimetres long and 18 millimetres high. The optic nerve at its entrance and exit can be seen. This specimen illustrates the fifth variety of this group.

9. In it the optic canal is almost completely surrounded by the posterior ethmoidal cell as it lies on the outer wall of the cell.

The cavity lies on its upper, inner, and lower walls and on part of its outer wall (*vide* Figure 10).

10. The inner wall of the optic canal lies against the posterior ethmoidal cell of that side.

Figure 12 illustrates this relationship in sagittal section, on the left side. The nerve is shown at its entry into the optic canal. The canal runs in the outer wall of the cell for a distance of 6 millimetres, and to demonstrate this the wall of the canal has been opened up.

11. The floor of the optic canal is formed on the one side by the roof of the posterior ethmoidal cell.

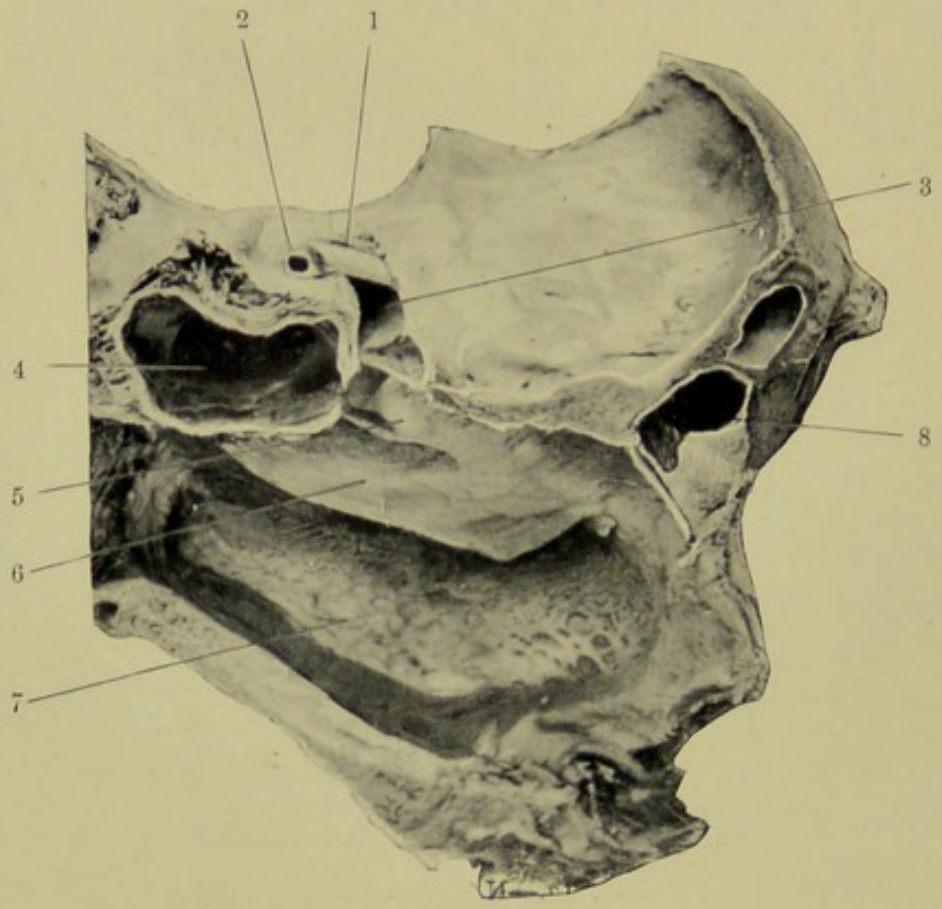


FIG. 13.—NATURAL SIZE.

1. Optic nerve.
2. Internal carotid artery.
3. Posterior ethmoidal cell.
4. Sphenoidal sinus.
5. Superior turbinal.
6. Middle turbinal.
7. Inferior turbinal.
8. Frontal sinus.

Figures 13 and 14 illustrate this. Figure 13 illustrates, in sagittal section, the course of the left optic nerve for a distance of 7 millimetres, as it passes above the posterior ethmoidal cell. Its bony covering has been removed.

12. On both sides the inner and lower walls of the optic canals, as well as the whole optic sulcus, are formed by the walls of the posterior ethmoidal cells.

In Figure 14 (Atlas) this relationship is shown, and also in a corre-

sponding sagittal section. Figure 15 illustrates this. The posterior ethmoidal cell in this specimen measures 22 millimetres in length, 38 millimetres in breadth, and 17 millimetres in height, on the right side. This cavity has expanded itself so as to extend over the sphenoidal sinuses, and lies thus in relation to both optic nerves and to

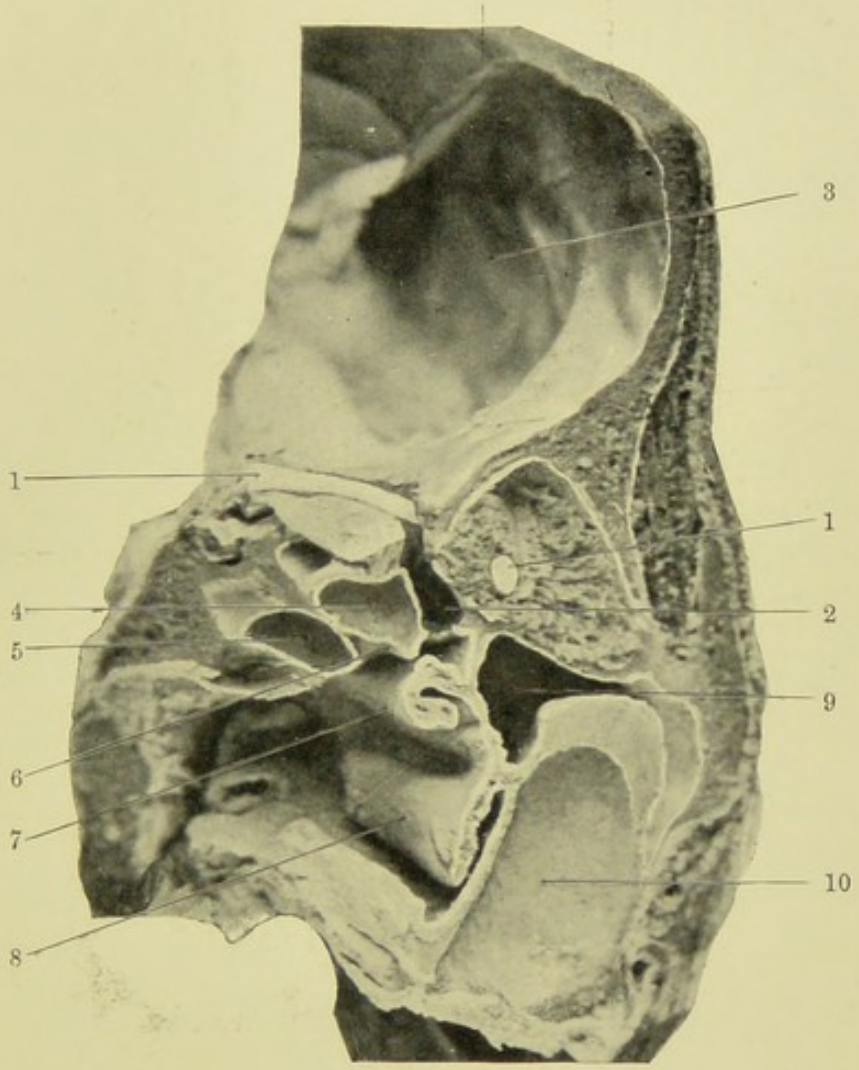


FIG. 14.—NATURAL SIZE.

1. Optic nerve.
2. Posterior ethmoidal cell.
3. Anterior cranial fossa.
4. Ethmoidal cell.
5. Sphenoidal sinus.
6. Superior turbinal.
7. Middle turbinal.
8. Inferior turbinal.
9. Maxillary antrum.
10. Alveolar cyst.

the chiasma. The floor of the optic canals and the whole optic sulcus in this manner are formed by a thin plate of bone, which separates it from the sinus.

13. The inner wall of the right optic canal is formed by the left posterior ethmoidal cell.

Figure 53 (Atlas) illustrates this in sagittal section. The sphenoidal sinuses are so asymmetrically placed that the left sphenoidal sinus lies in front of the right. The right sinus is 18 millimetres long,

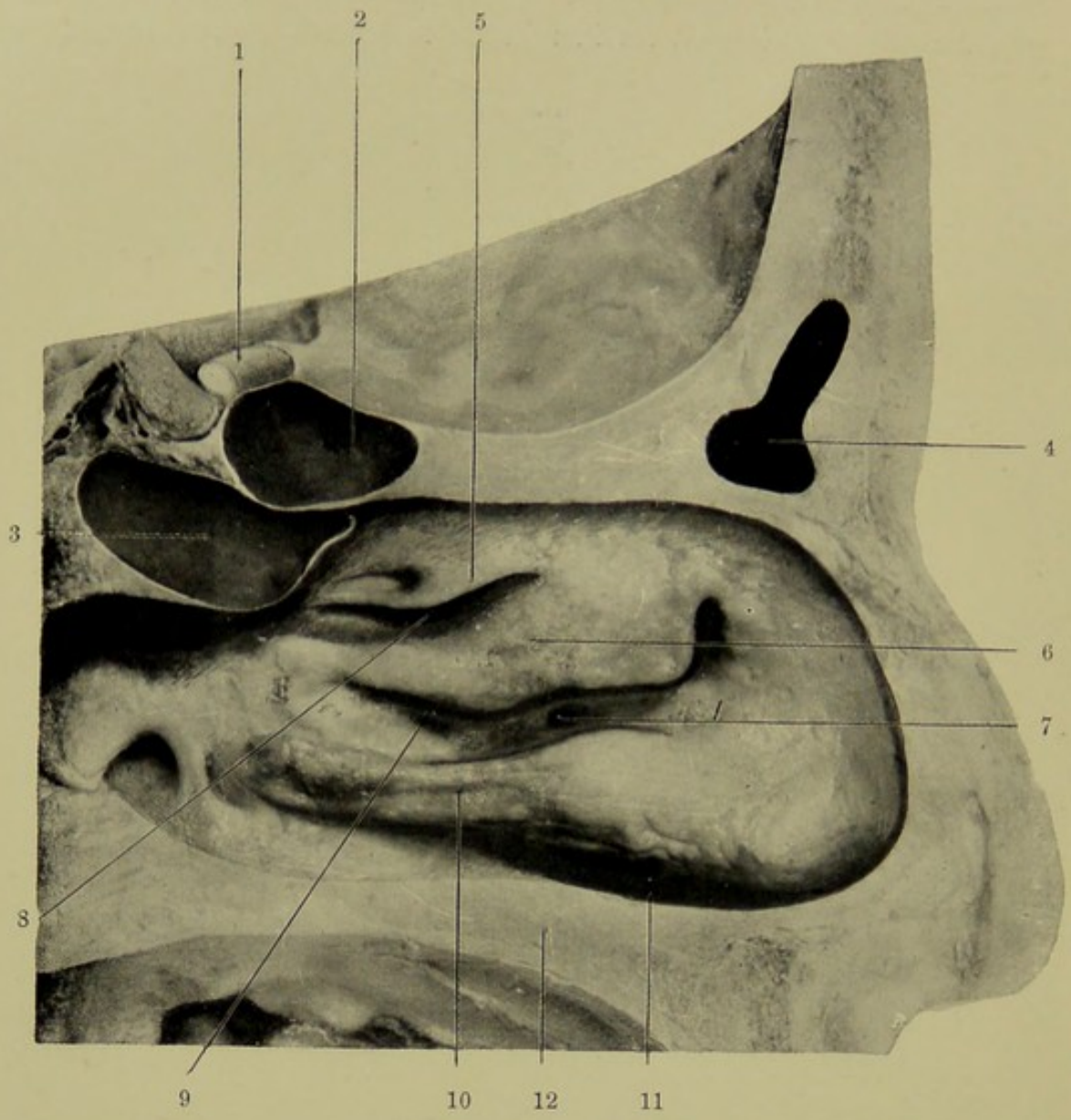


FIG. 15.—NATURAL SIZE.

1. Left optic nerve.
2. Posterior ethmoidal cell.
3. Left sphenoidal sinus.
4. Left frontal sinus.
5. Superior turbinal.
6. Middle turbinal.
7. Ostium of maxillary antrum.
8. Superior meatus.
9. Middle meatus.
10. Inferior turbinal.
11. Inferior meatus.
12. Palate.

15 millimetres broad, and 16 millimetres high; the left is 15 millimetres long, 16 millimetres broad, and 17 millimetres high. The left posterior ethmoidal cell is 16 millimetres long and 15 millimetres broad.

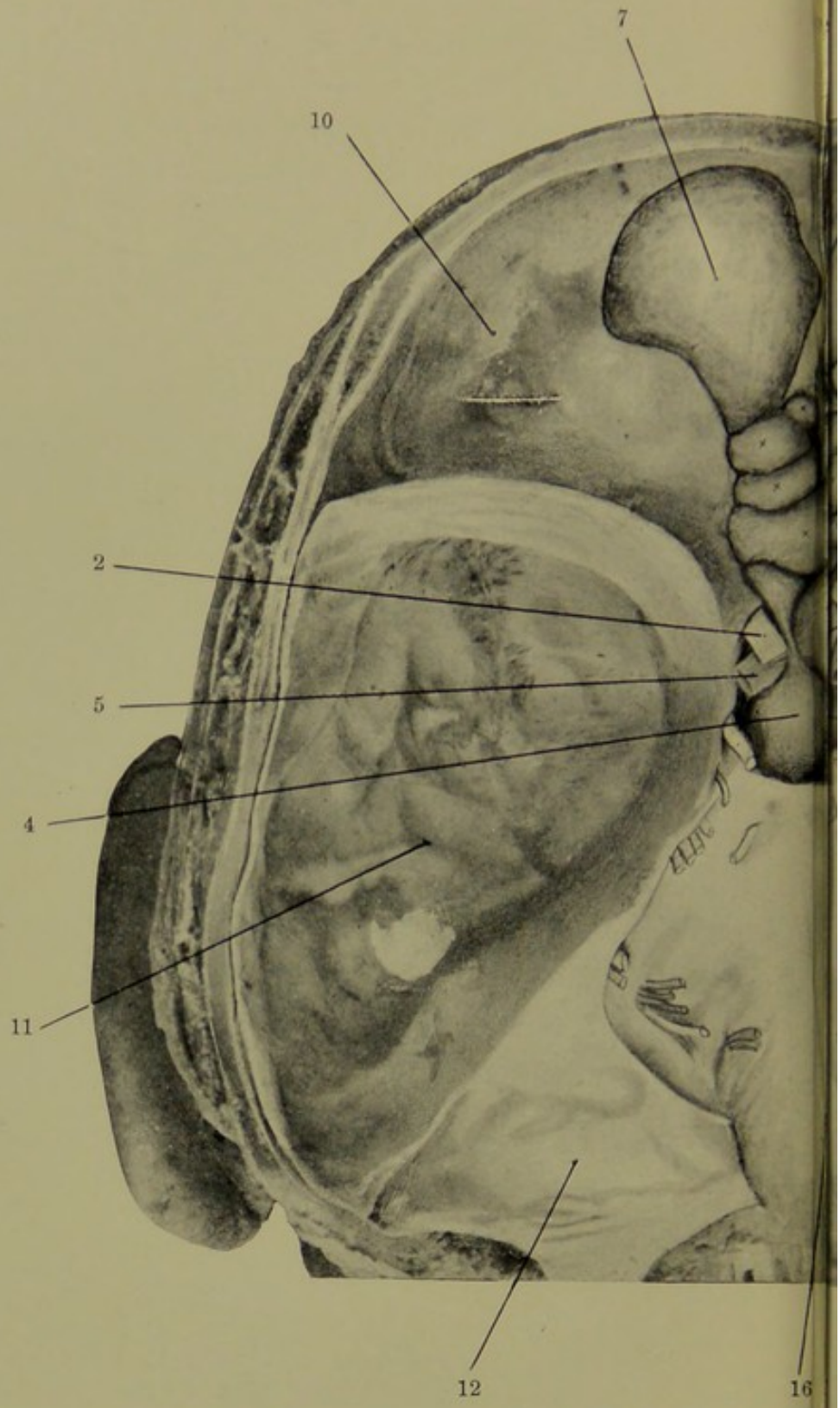
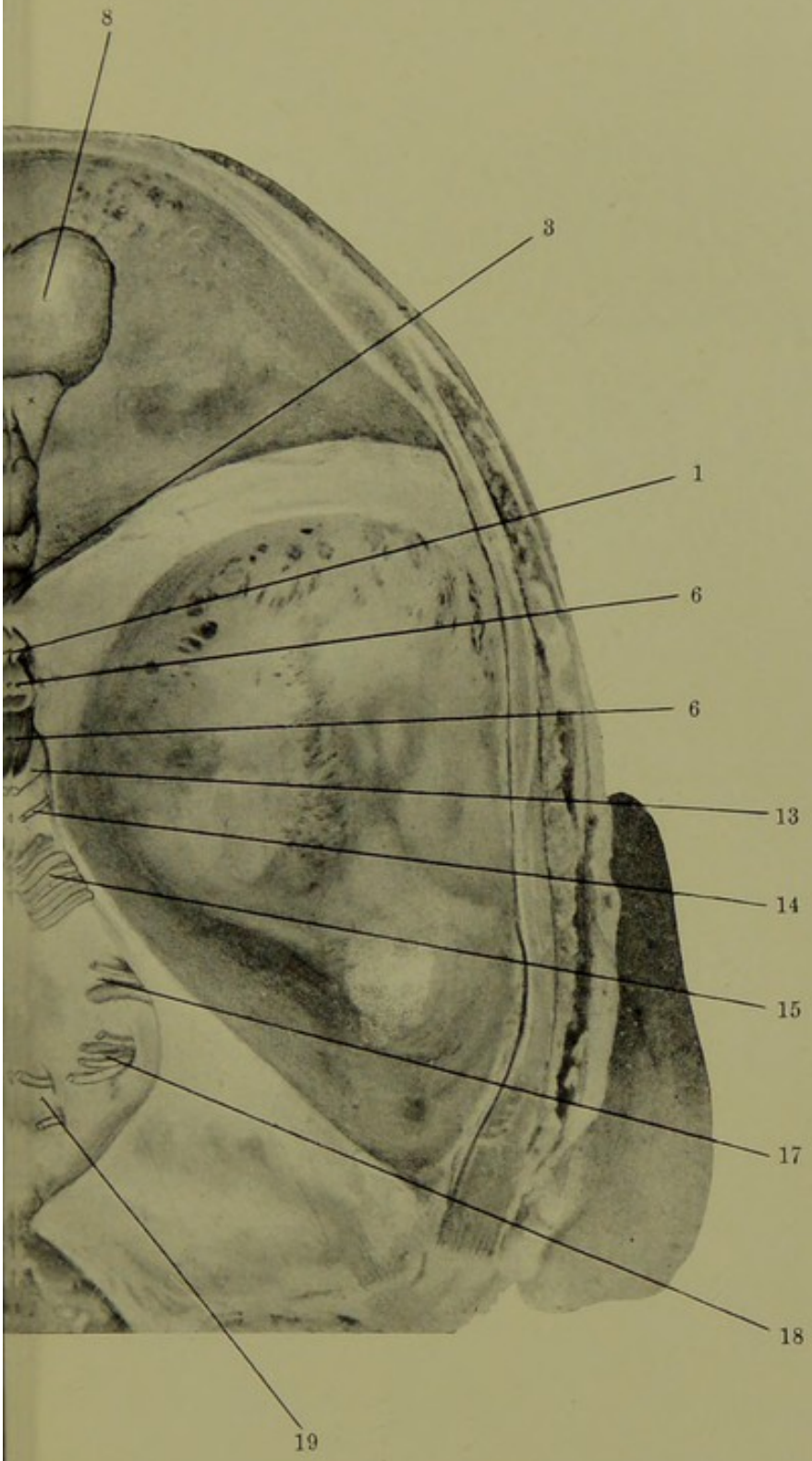


FIG. 16.—N

1. Right optic nerve.
2. Left optic nerve.
3. Right sphenoidal sinus.
4. Left sphenoidal sinus.
5. Left internal carotid artery.
6. Right internal carotid artery.
7. Left frontal sinus.
8. Right frontal s
9. Olfactory fissu
10. Anterior crani
11. Middle cranial
12. Posterior cran
13. Third nerve—
14. Fourth nerve—



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15. Fifth nerve—trigeminal.
16. Sixth nerve—abducens.
17. Facial and auditory nerves.
18. Glosso-pharyngeal nerve, vagus nerve, spinal accessory nerve.
19. Hypoglossal nerve.

× × × × × × × × ×, Ethmoidal cells.

Group 4.

On both sides the optic nerves stand in close relation to the sphenoidal sinuses. There are five varieties.

14. On both sides the inner and lower walls of the optic canals and the wall of the optic sulcus are formed by the sphenoidal sinuses. In Figure 16, which illustrates a specimen hardened in formalin,

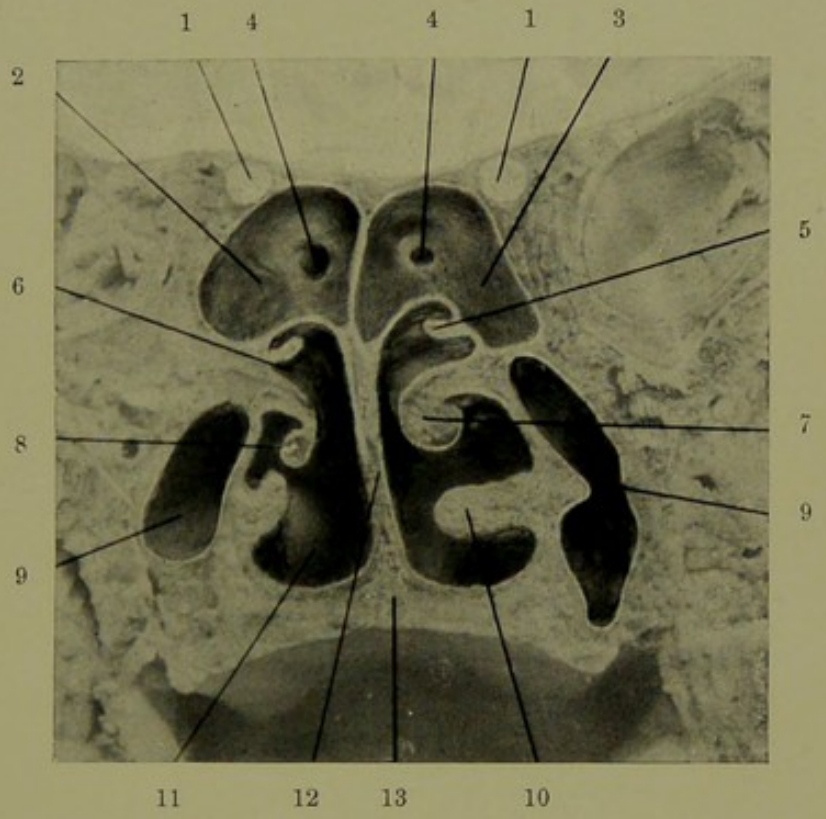


FIG. 17.—NATURAL SIZE.

1. Optic nerve.
2. Right sphenoidal sinus.
3. Left sphenoidal sinus.
4. Sphenoidal ostium.
5. Superior turbinal.
6. Superior meatus.
7. Middle turbinal.
8. Middle meatus.
9. Maxillary antrum.
10. Inferior turbinal.
11. Inferior meatus.
12. Septum.
13. Palate.

it can be seen that the upper walls of the accessory sinuses have been removed through the anterior and middle cranial fossæ. The mucous membranes, in their original saccular form, are seen seriatim from before backward. They are the lining membranes of the frontal, ethmoidal, and sphenoidal sinuses. The right sphenoidal sinus lies in front of the left, and the left stretches back behind the right.

15. On both sides the inner walls of the optic canal and the wall of the corresponding third of the optic sulcus are formed by the sphenoidal sinuses.

Figure 75 (Atlas) illustrates in frontal section the sphenoidal sinuses, with the optic nerves above them, as they enter the optic canals.

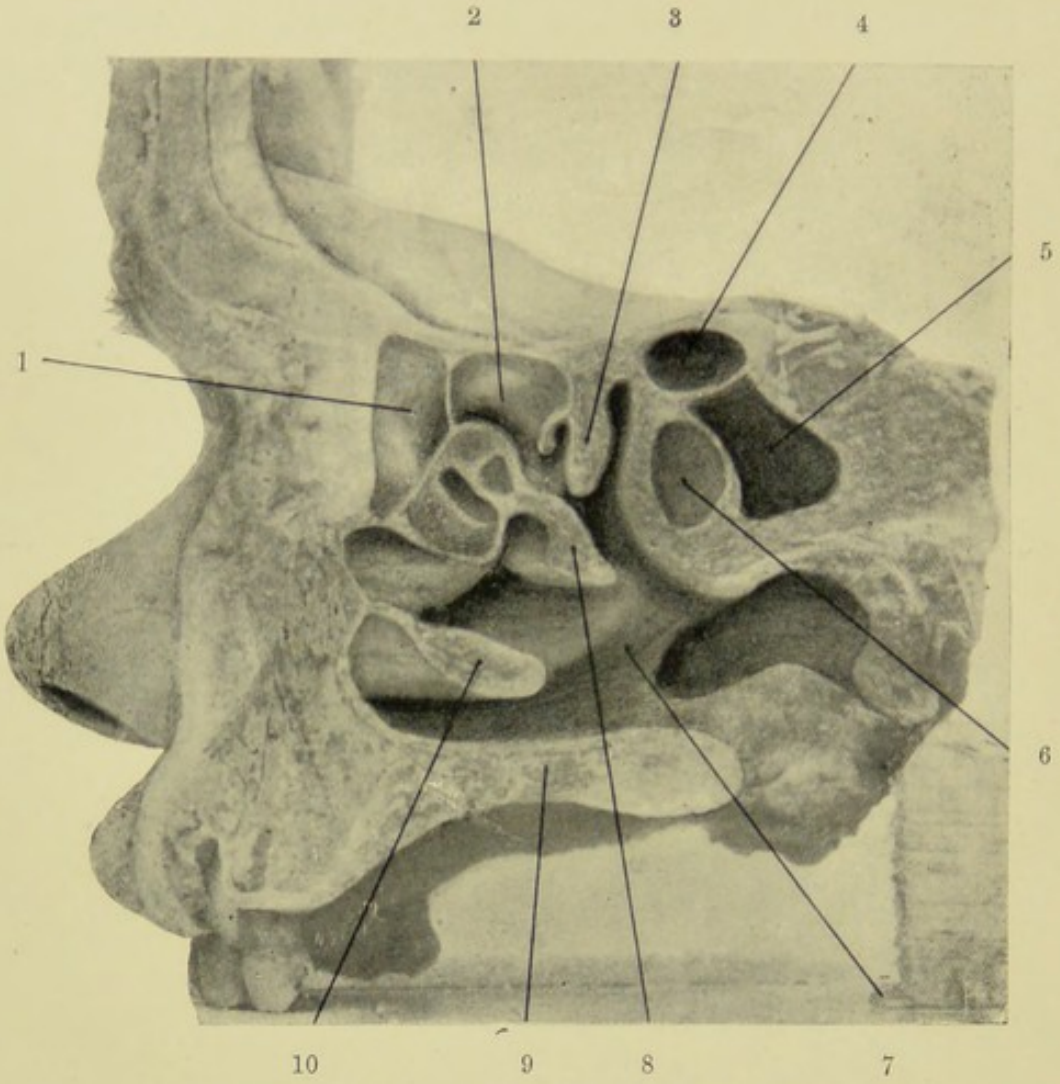


FIG. 18.—NATURAL SIZE.

1. Anterior ethmoidal cell.
2. Posterior ethmoidal cell.
3. Superior turbinal.
4. Posterior ethmoidal cell—left.
5. Right sphenoidal sinus.
6. Left sphenoidal sinus.
7. Septum.
8. Middle turbinal.
9. Palate.
10. Inferior turbinal.

Figure 17 illustrates the same relationship. The sphenoidal sinuses in this are 17 millimetres broad, 28 millimetres long, and 22 millimetres high on the right, and on the left 17 millimetres broad, 25 millimetres long, and 22 millimetres high.

Figure 18 shows the accessory sinuses and nasal passages in sagittal section. The sphenoidal sinuses are asymmetrical, the left sinus (6)

lying in front of the right (5). The right sinus is 18 millimetres long, 15 millimetres broad, and 16 millimetres high; the left sinus is 15 millimetres long, 16 millimetres broad, and 17 millimetres high. Immediately above the right sphenoidal sinus is the most posterior ethmoidal cell; it is 16 millimetres long, 15 millimetres broad, and 15 millimetres high.

Figure 19 shows in frontal section the optic nerves in this relation to the lateral walls of the sphenoidal sinuses.

16. On each side the inferior wall of the optic canal and the wall of the optic sulcus are formed by the sphenoidal sinus. Figure 19

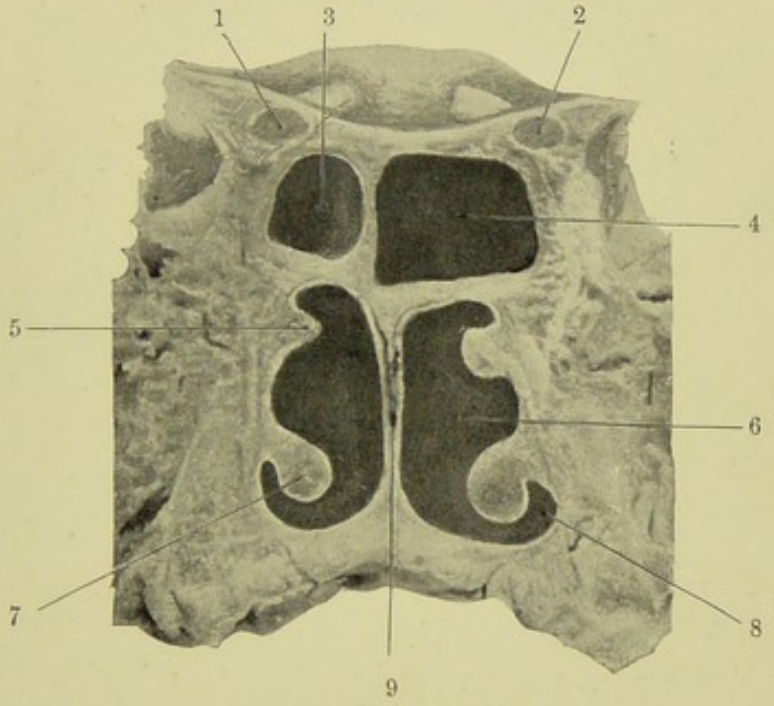


FIG. 19.—NATURAL SIZE.

1. Right optic nerve.
2. Left optic nerve.
3. Right sphenoidal sinus.
4. Left sphenoidal sinus.
5. Middle turbinal.
6. Middle meatus.
7. Inferior turbinal.
8. Inferior meatus.
9. Septum.

illustrates this relationship. The left sphenoidal sinus shows, however, a greater expansion, and forms the floor of the optic canal as well as both the middle and left third of the optic sulcus.

17. The wall of the optic sulcus on both sides is formed by the sphenoidal sinuses.

18. On the right side the inner wall of the optic canal and the optic sulcus are formed by the sphenoidal sinus on that side, and on the left side the inferior wall of the optic canal is formed by the left sphenoidal sinus.

Figure 20 shows in a vertical transverse section the close relation of the optic nerves to the sphenoidal sinuses on both sides.

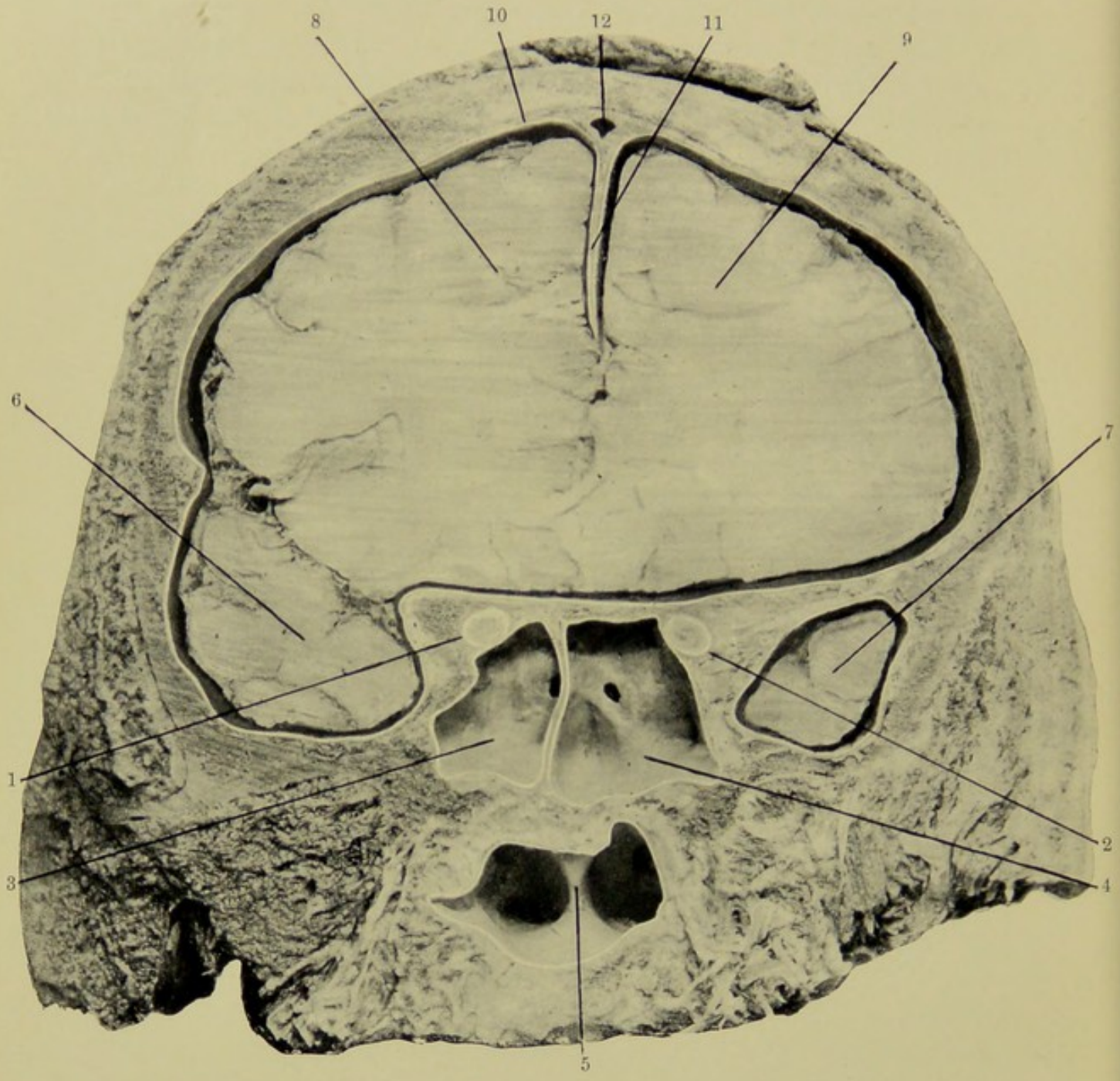


FIG. 20.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3. Left sphenoidal sinus.
4. Right sphenoidal sinus.
5. Septum.
6. Left temporal lobe.
7. Right temporal lobe.
8. Left frontal lobe.
9. Right frontal lobe.
10. Dura mater.
11. Falx cerebri.
12. Superior longitudinal sinus.

Group 5.

The optic nerves in this class show seven varieties of relationship to the sphenoidal sinuses, four of which are to the sinus of the same side, two to that of the opposite side, and in one the sinus lies in the middle line.

19. The lower wall of the optic canal on the left side is formed by the wall of the left sphenoidal sinus, and also the whole optic sulcus is formed by the left sphenoidal sinus.

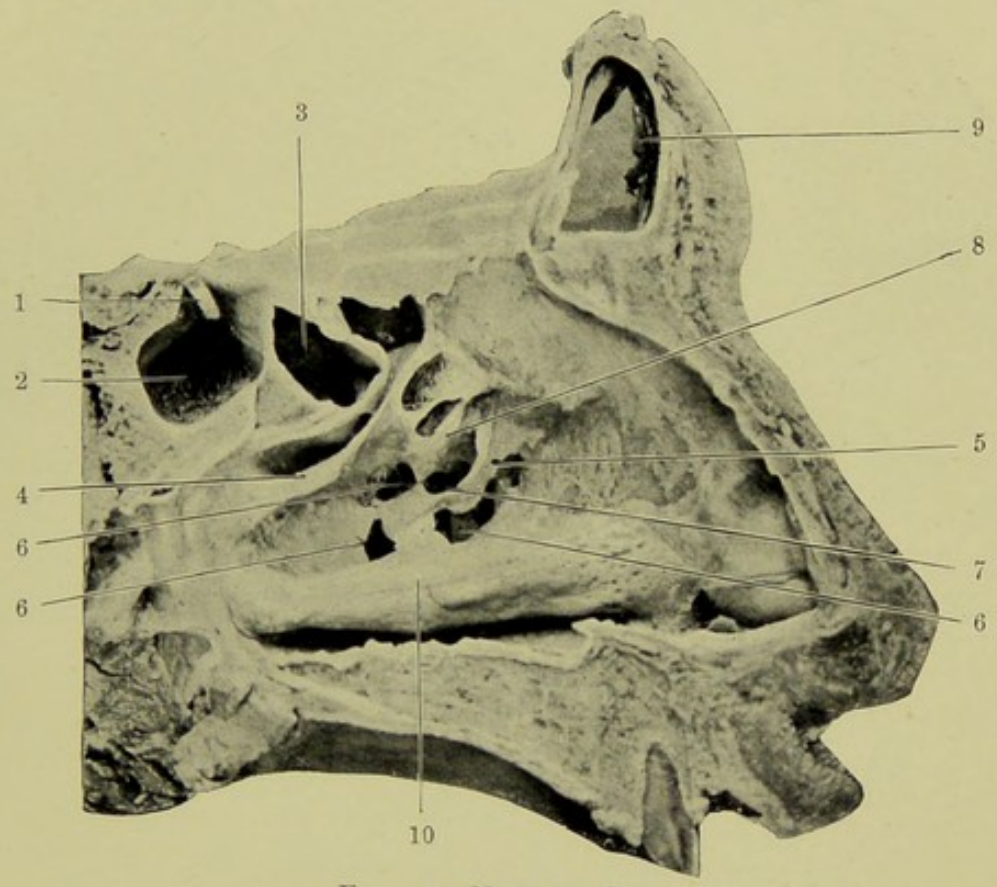


FIG. 21.—NATURAL SIZE.

1. Optic nerve.
2. Sphenoidal sinus.
3. Posterior ethmoidal cell.
4. Middle turbinal.
5. Unciform process.
6. Fontanelles of the antrum, where the bony wall is defective.
7. Ostium of maxillary antrum.
8. Ethmoidal bulla.
9. Frontal sinus.
10. Inferior turbinal.

20. The left sphenoidal sinus forms the floor of the left optic canal and the wall of the left third of the optic sulcus. A similar relationship may occur on the right side.

21. The sphenoidal sinus on the right side forms the inferior wall of the right optic canal. Figure 21 illustrates a formalin-hardened specimen, in which the bone walls of the right orbital cavity have

been removed, and also those of the ethmoidal cells, that are in relation to the optic nerve. This relation of the optic nerve to the sphenoidal sinuses and ethmoidal cells is here well illustrated.

Figure 4, also a formalin-hardened specimen, shows the intimate relation between the left optic nerve and the left sphenoidal sinus.

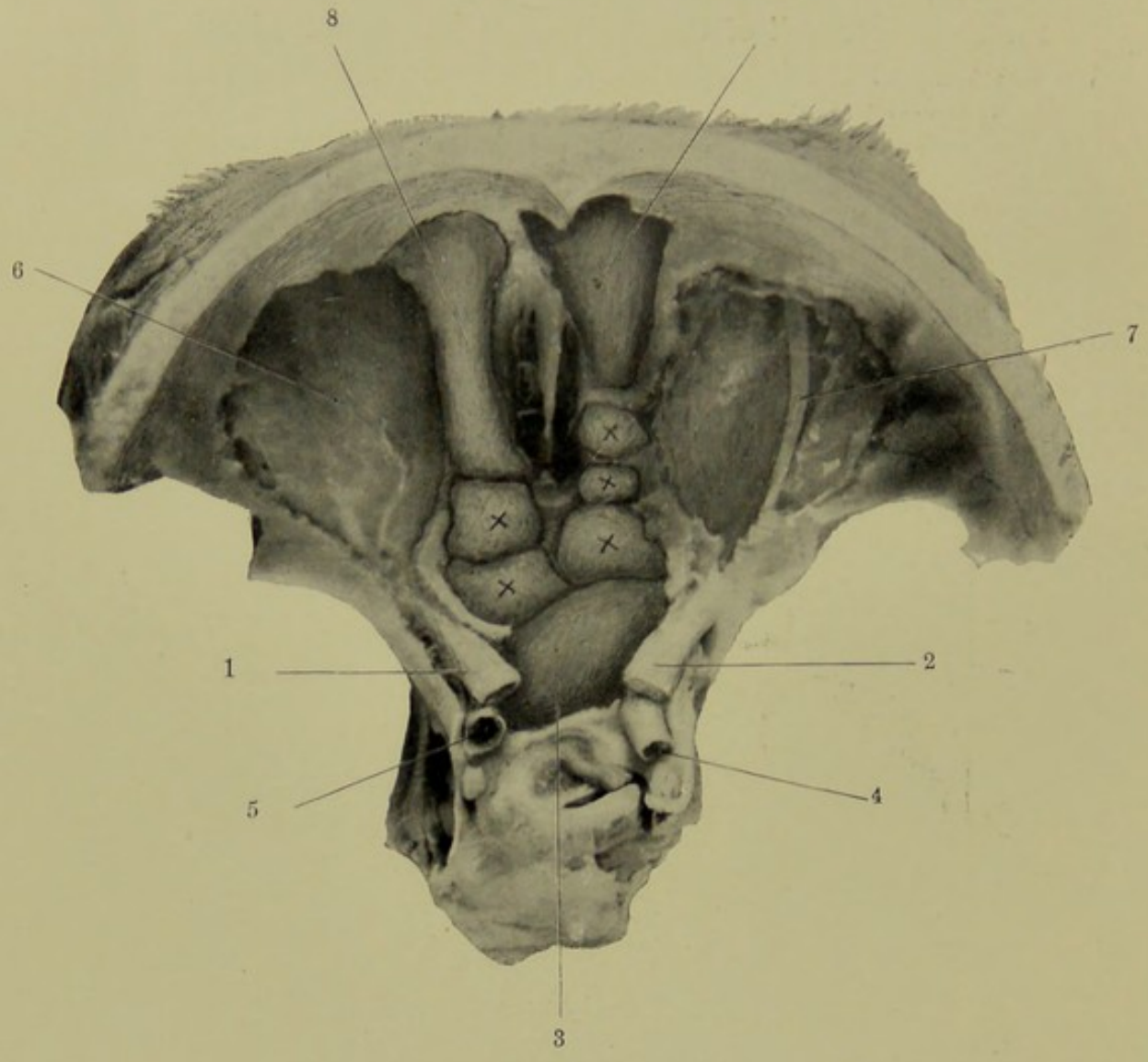


FIG. 22.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3. Right sphenoidal sinus.
4. Right internal carotid artery.
5. Left internal carotid artery.
6. Orbit.
7. Supra-orbital nerve.
8. Left frontal sinus.
9. Right frontal sinus.

This sinus is here 12 millimetres long, and is separated from the floor of the optic canal only by a very thin plate of bone. Both the chiasma and the right optic nerve are separated from the sinus by thick, spongy bone.

22. The right sphenoidal sinus forms the medial wall of the right

optic canal. This is shown in Figure 21, in sagittal section. The thin septum between the optic canal and the sphenoidal sinus has been removed, and the course of the optic nerve, 5 millimetres long, can be seen on the side-wall of the sinus.

23. The medial wall and floor of both optic canals and the whole wall of the optic sulcus are formed by the right sphenoidal sinus. Figures 22 and 23 demonstrate this.

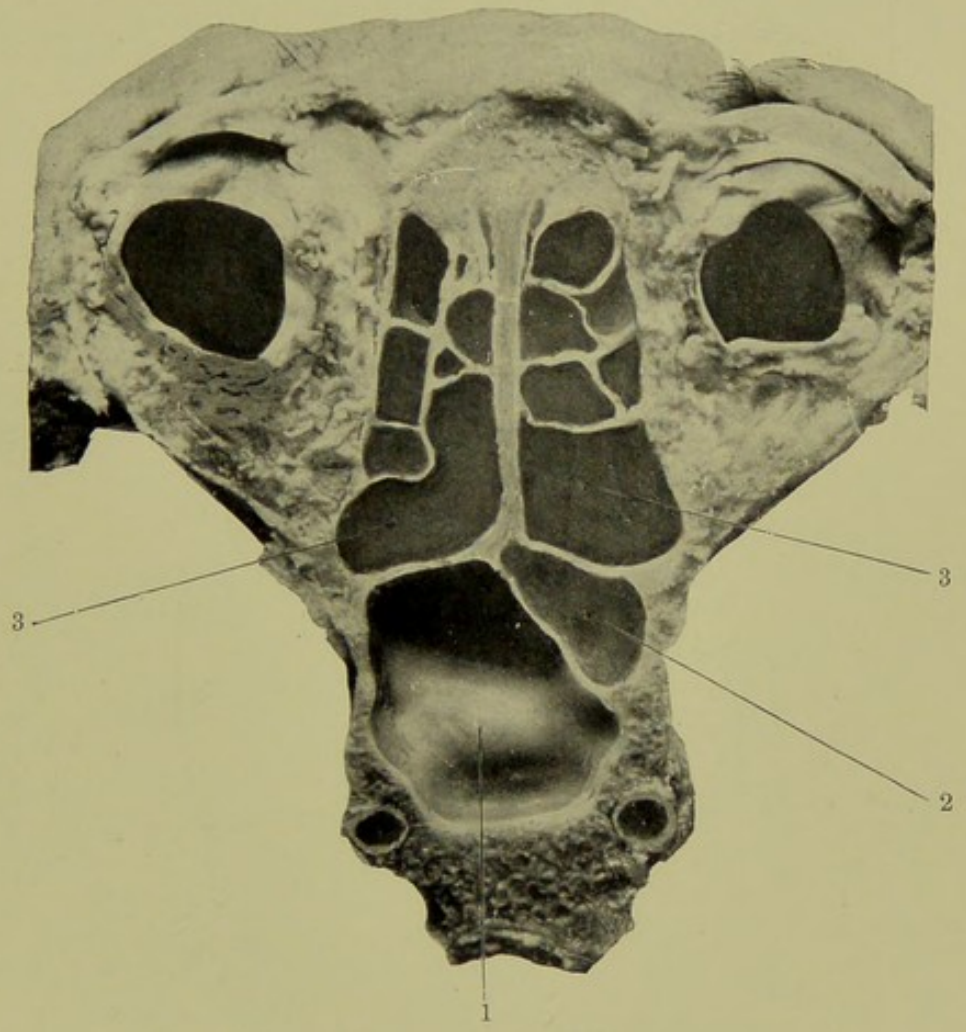


FIG. 23.—NATURAL SIZE.

1 and 2. Sphenoidal sinuses. 3, 3. Posterior ethmoidal cell.

Figure 22, a formalin-hardened specimen, shows from above the intimate relations of the right sphenoidal sinus to both optic nerves, while Figure 23 shows the exceptionally marked asymmetry between the two sphenoidal sinuses, the left one being of small size, and pushed quite laterally.

24. The right sphenoidal sinus, as is seen in Figure 24, is in intimate

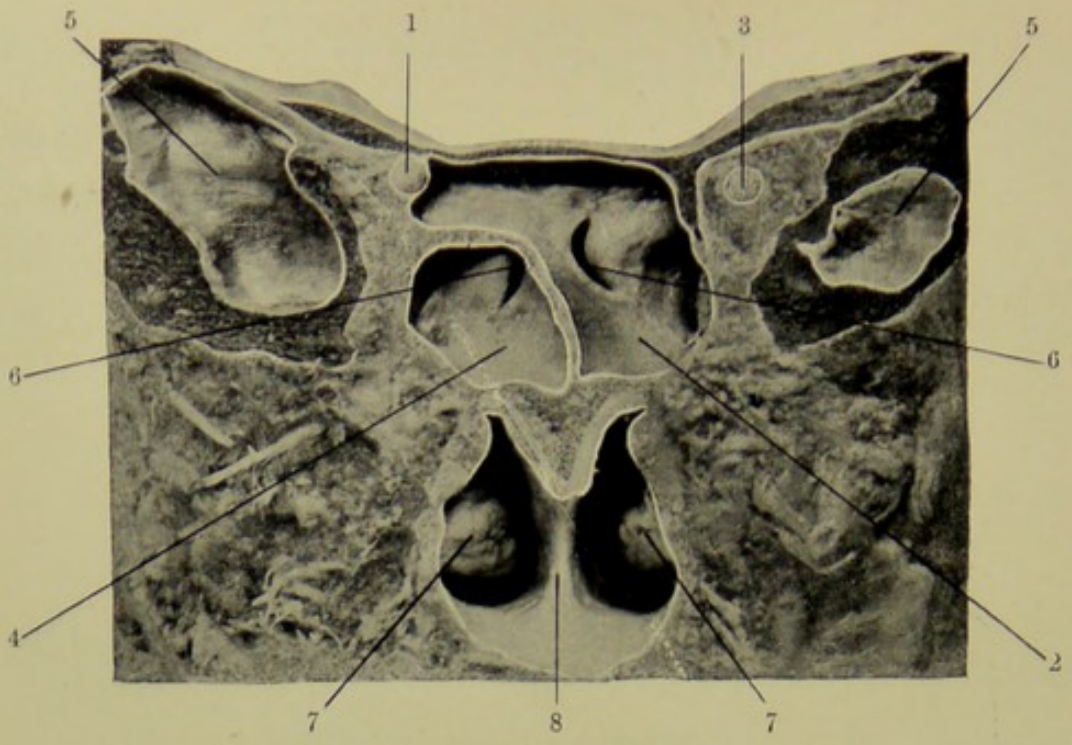


FIG. 24.—NATURAL SIZE.

1. Left optic nerve.
2. Right sphenoidal sinus.
3. Right optic nerve.
4. Left sphenoidal sinus.
5. Middle cranial fossa.
6. Sphenoidal ostium.
7. Inferior turbinal—posterior end.
8. Septum—posterior edge.

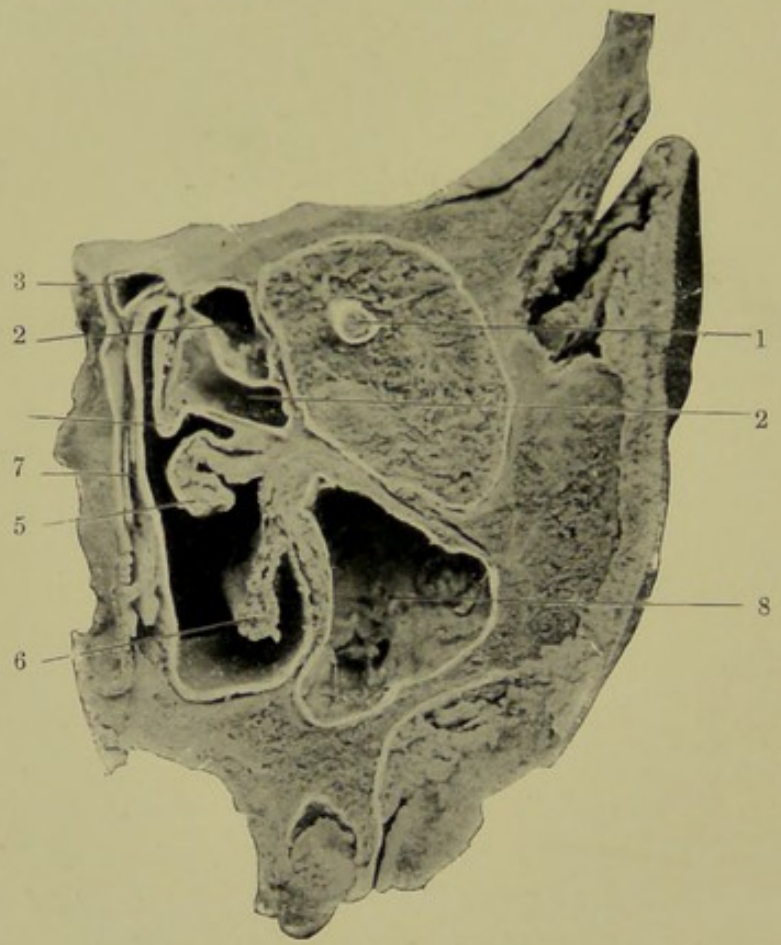


FIG. 25.—NATURAL SIZE.

1. Optic nerve.
2. Ethmoidal cell.
3. Left sphenoidal sinus.
4. Superior turbinal.
5. Middle turbinal.
6. Inferior turbinal.
7. Septum.
8. Maxillary antrum.

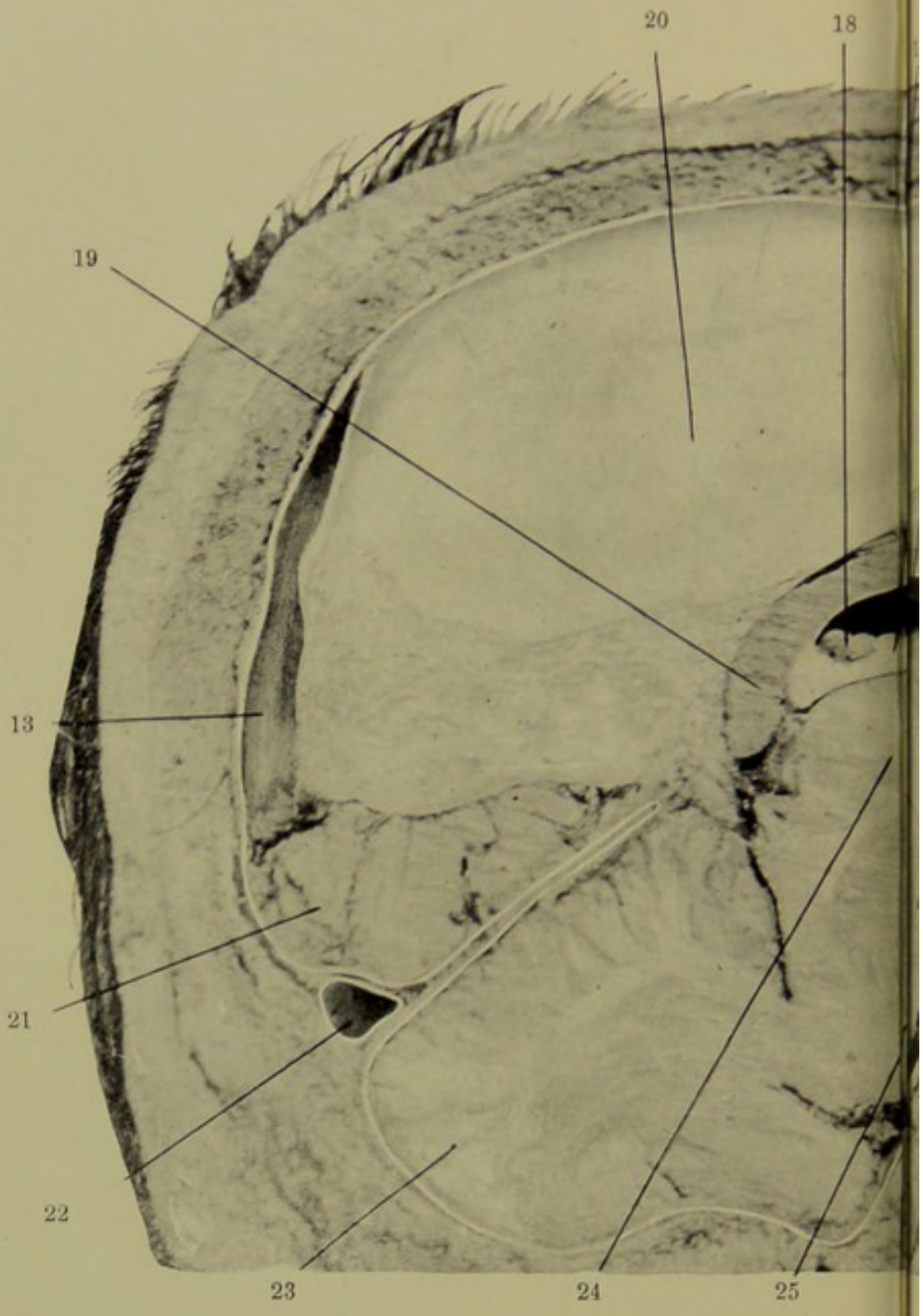
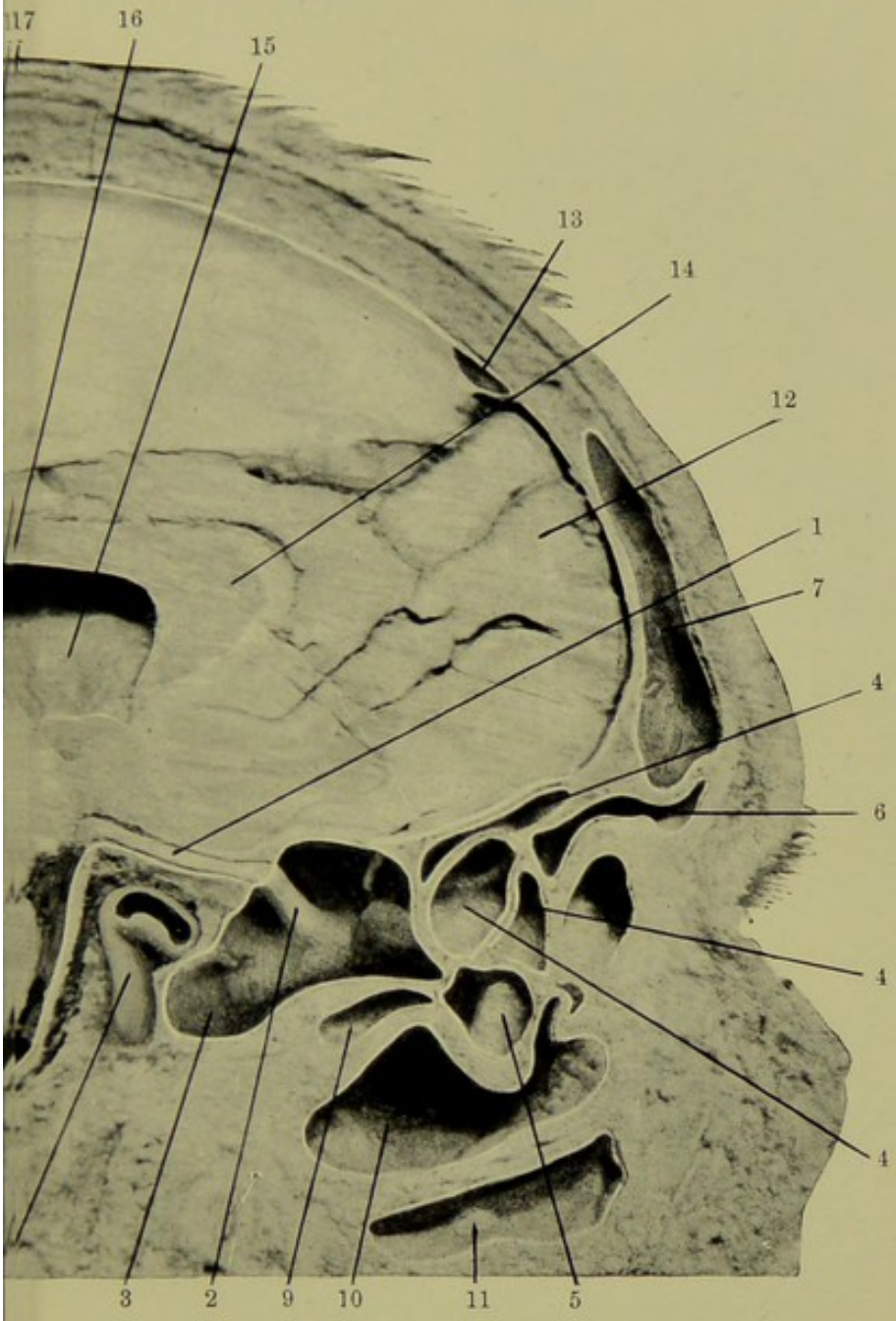


FIG. 26.—

1. Optic nerve.
2. Optic canal.
3. Sphenoidal sinus.
4. Anterior ethmoidal cell.
5. Ethmoidal bulla.
6. Left frontal sinus.
7. Right frontal sinus.
8. Internal carotid artery.
9. Superior meatus.
10. Middle meatus.
11. Inferior meatus.
12. Left frontal lobe.
13. Superior longitudinal sinus.



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14. Genu of corpus callosum.
15. Caudate nucleus.
16. Lateral ventricle.
17. Fornix.
18. Choroid plexus.
19. Splenium of corpus callosum.
20. Falciform process.
21. Occipital lobe.
22. Straight sinus.
23. Cerebellum.
24. Optic thalamus.
25. Pons.

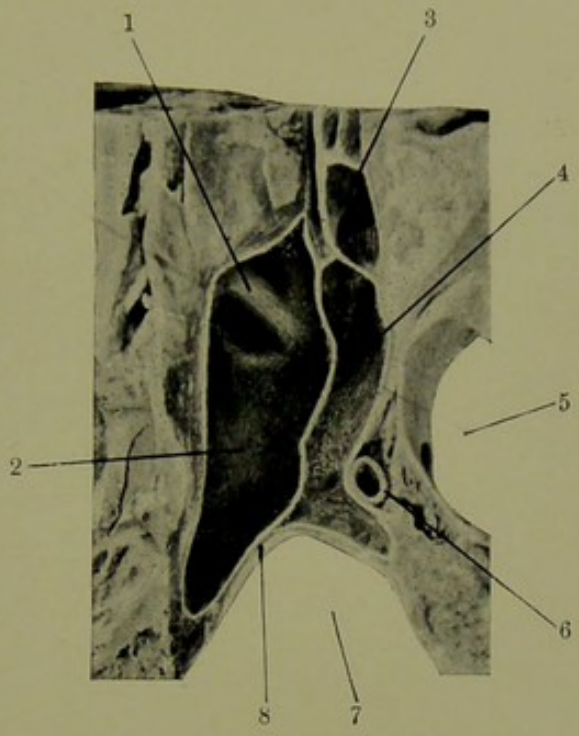


FIG. 27.—NATURAL SIZE.

1. Left optic canal.
2. Left sphenoidal sinus.
3. Right posterior ethmoidal cell.
4. Right sphenoidal sinus.
5. Middle cranial fossa.
6. Internal carotid artery.
7. Posterior cranial fossa.
8. Clivus.

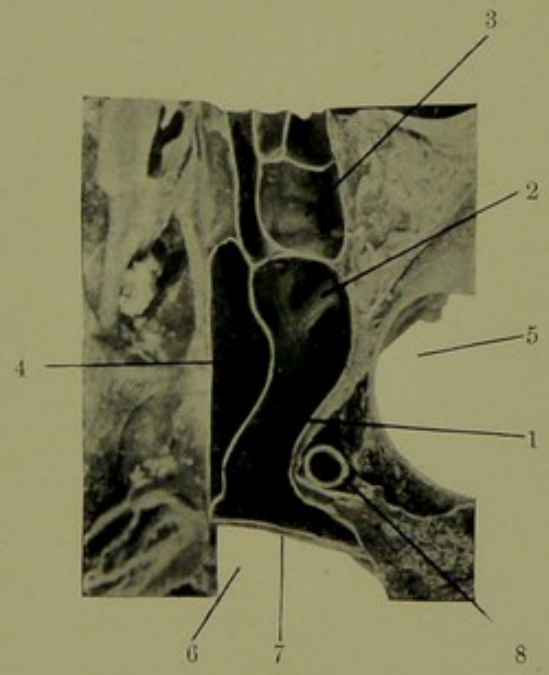


FIG. 28.—NATURAL SIZE.

1. Right sphenoidal sinus.
2. Right optic canal.
3. Posterior ethmoidal cell.
4. Left sphenoidal sinus.
5. Middle cranial fossa.
6. Posterior cranial fossa.
7. Clivus.
8. Internal carotid artery.

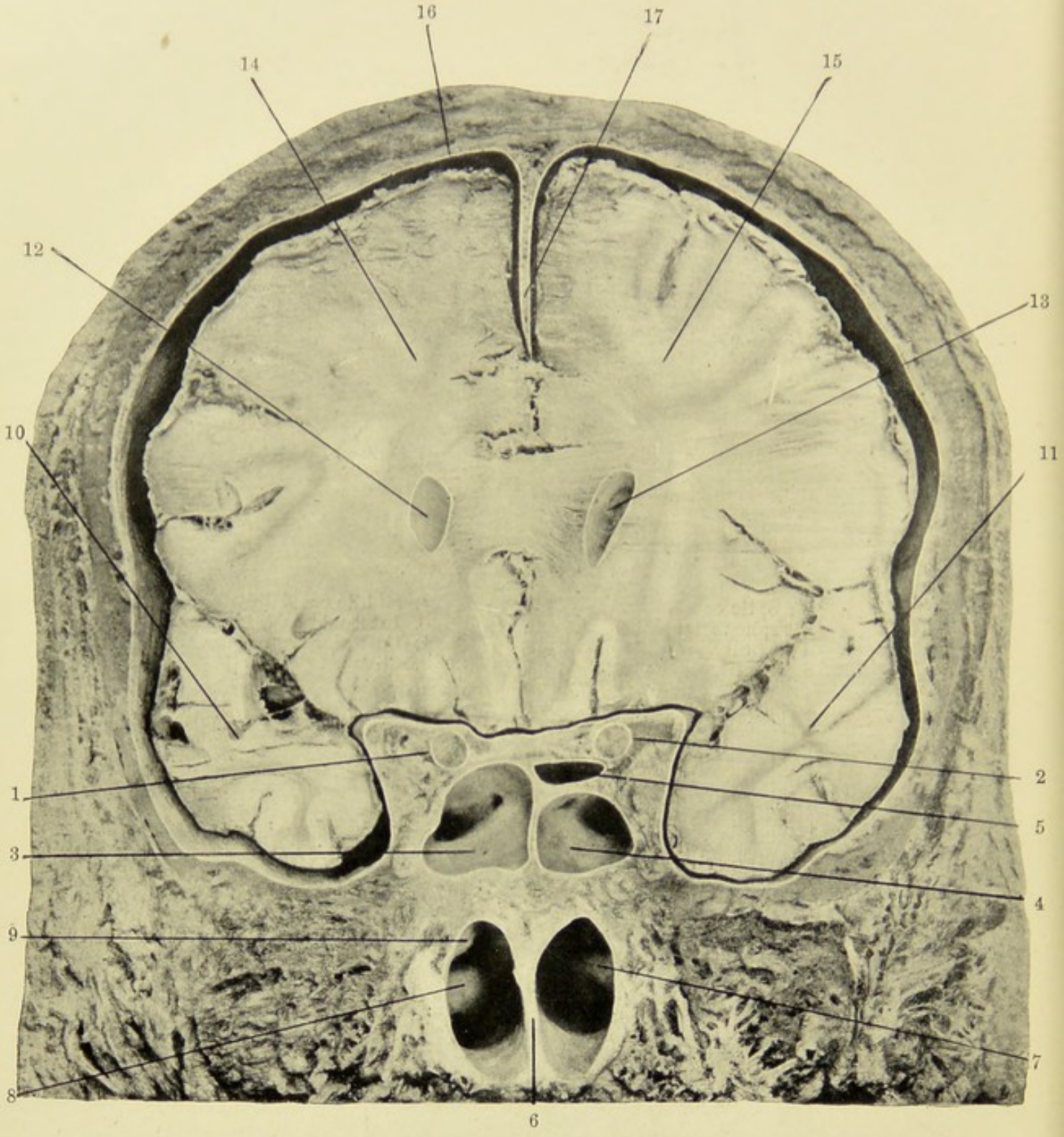


FIG. 29.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3. Left sphenoidal sinus.
4. Right sphenoidal sinus.
5. Right posterior ethmoidal cell.
6. Septum.
7. Right inferior turbinal—posterior end.
8. Left inferior turbinal—posterior end.
9. Left middle turbinal—posterior end.
10. Left temporal lobe.
11. Right temporal lobe.
12. Left lateral ventricle.
13. Right lateral ventricle.
14. Left frontal lobe.
15. Right frontal lobe.
16. Dura mater.
17. Falciform process.

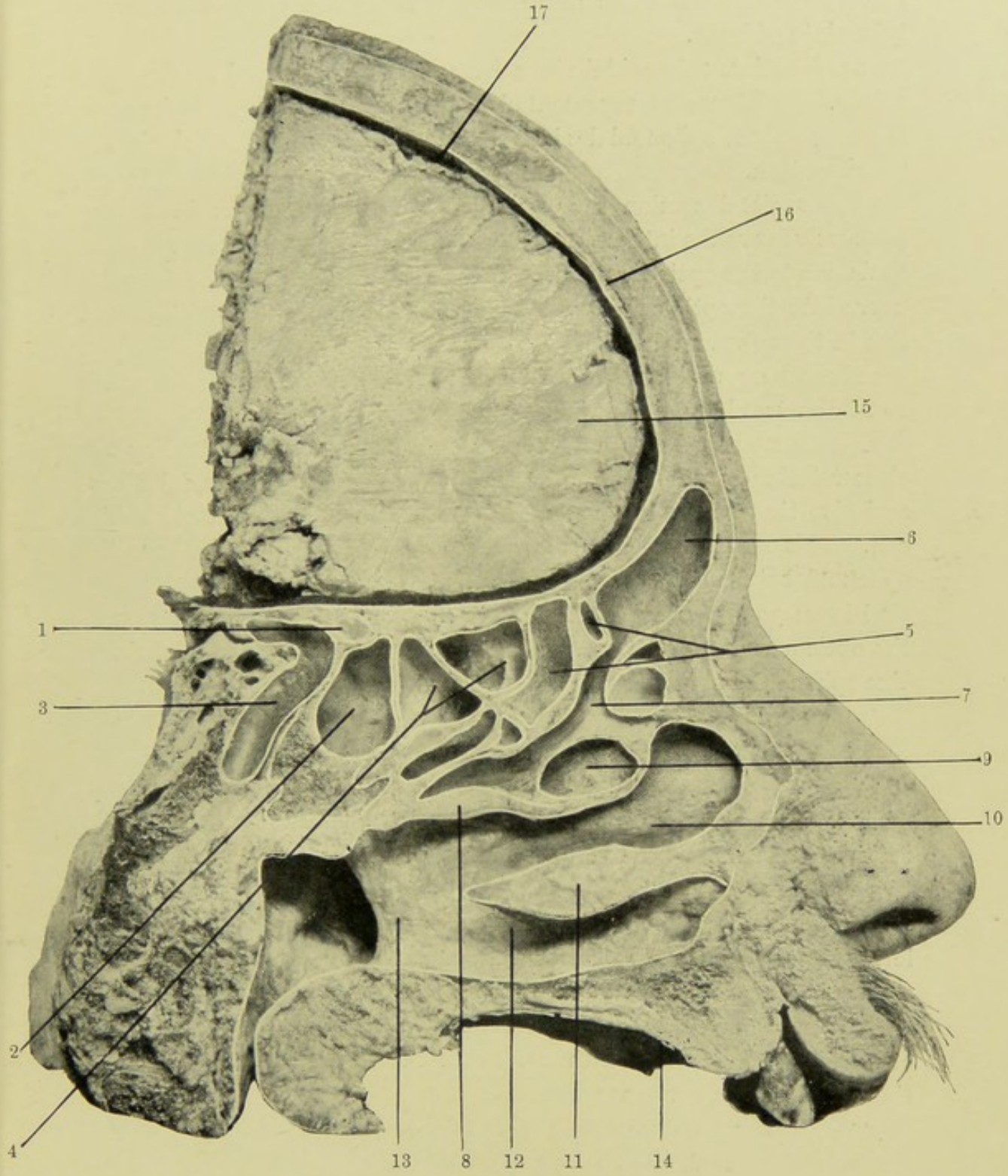


FIG. 30.—NATURAL SIZE.

1. Optic nerve.
2. Sphenoidal sinus.
3. Internal carotid artery.
4. Posterior ethmoidal cell.
5. Anterior ethmoidal cell.
6. Frontal sinus.
7. Naso-frontal duct.
8. Middle turbinal.
9. Bone-cell of the middle turbinal.
10. Middle meatus.
11. Inferior turbinal.
12. Inferior meatus.
13. Septum.
14. Palate.
15. Frontal lobe.
16. Dura mater.
17. Arachnoid.

relation to the left optic nerve, and is separated from it by a very thin plate of bone.

25. The left sphenoidal sinus forms the middle third of the optic sulcus. In Figure 25, represented in frontal section, this can be seen. Here the left sphenoidal sinus lies in the middle line, above the nasal septum.

The sagittal section represented in Figure 26 shows in a very instructive way the intracranial course of the optic nerve from the optic thalamus to the optic foramen. It also shows the entrance of the optic nerve into the optic canal, which runs for a distance of 10 millimetres free in the sphenoidal sinus.

Figures 27 and 28 show in horizontal section the optic canal, 10 millimetres long, running free in the sphenoidal sinus.

Figure 29 represents a vertical transverse section illustrating the close relation of the right optic nerve to the right sphenoidal sinus, and also that of the left optic nerve to the most posterior ethmoidal cell. This most posterior cell on the left side is situated above the sphenoidal sinus.

Figure 30 shows a sagittal section illustrating the close relation of the optic nerves and the internal carotid artery to the sphenoidal sinus. The sphenoidal sinus forms the thin lower wall of the optic canal.

Group 6.

The most posterior ethmoidal cell and the sphenoidal sinus are in intimate relation with the optic nerve of the same side. There are two varieties.

26. The left posterior ethmoidal cell forms the medial wall, and the left sphenoidal sinus the inferior wall, of the left optic canal. Figure 5, a formalin-hardened specimen, illustrates this.

The nasal cavities are opened from without. The most posterior ethmoidal cell, marked with a **X**, and the sphenoidal sinus in relation to the optic nerve are seen.

27. The left most posterior ethmoidal cell forms the medial wall of the left optic canal, and the left sphenoidal sinus forms the floor of the right optic canal and the wall of the right and middle third of the optic sulcus. Figure 31, a formalin-hardened specimen, illustrates this. In it the inner wall of the orbital cavity has been removed, and the mucous membrane of the accessory sinuses is shown in saccular form.

The most posterior ethmoidal cell on the left side, marked with a X, in relation to the left optic nerve, and the left sphenoidal

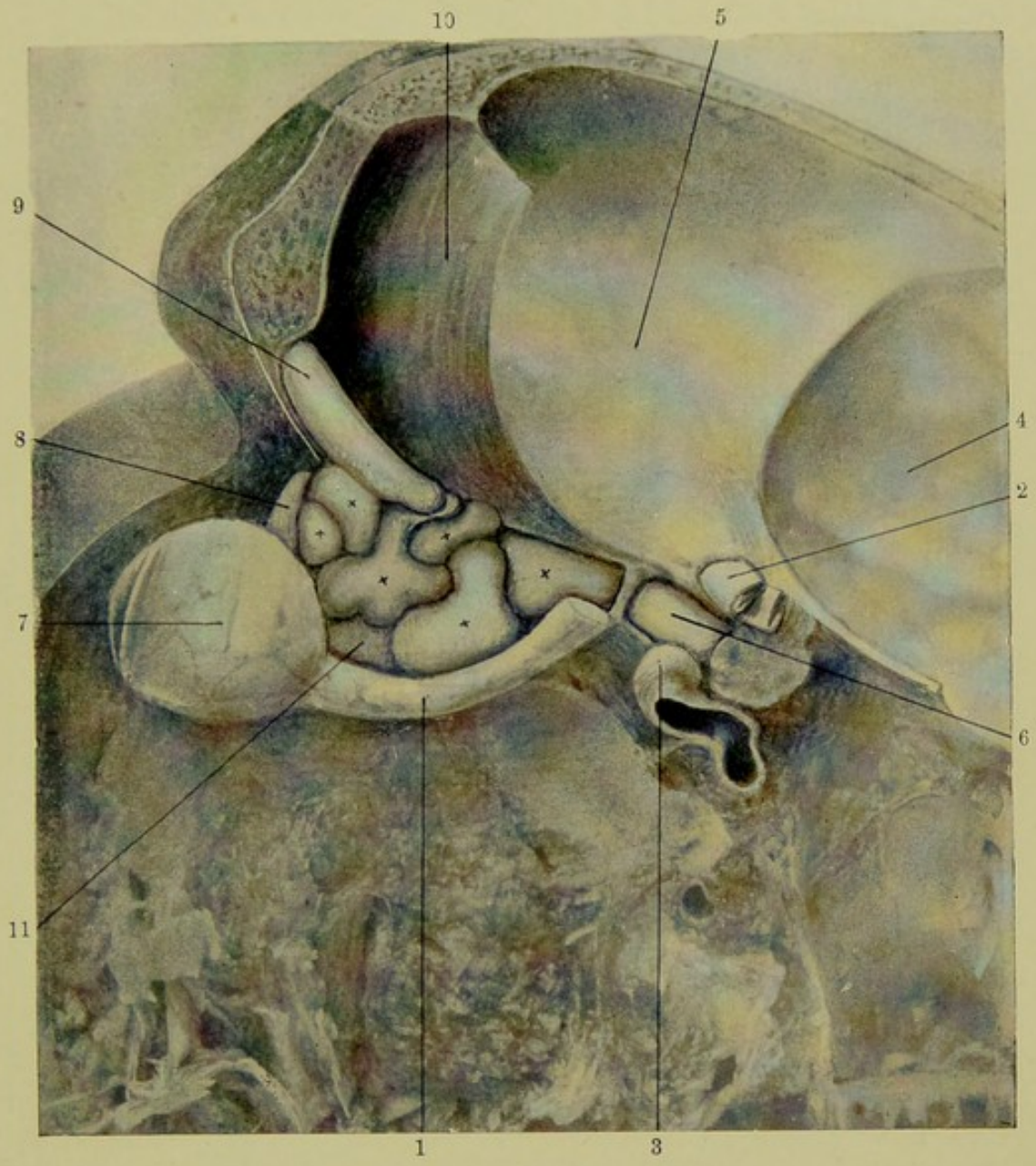


FIG. 31.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3. Internal carotid artery.
4. Middle cranial fossa.
5. Anterior cranial fossa.
6. Sphenoidal sinus—left.
7. Eyeball.
8. Lachrymal sac.
9. Frontal sinus.
10. Falciform process.
11. Maxillary sinus.

× × × × × ×, Ethmoidal cells—anterior and posterior.

sinus in its relation to the right optic nerve, are shown. The same relationship, in the absence of the left sphenoidal sinus, may occur on the right side.

Group 7.

The posterior ethmoidal cells and the sphenoidal sinuses show a similar relationship on both sides to the optic nerves.

28. The most posterior ethmoidal cells form on both sides the medial wall of the optic canal, and the sphenoidal sinuses form the wall of the optic sulcus.

Group 8.

The most posterior ethmoidal cells on both sides and the sphenoidal sinus of one side are in relation to the optic nerves. There are two varieties.

29. The right posterior cell forms the floor and medial wall of the right optic canal, and the wall of the right third of the optic sulcus. The left posterior ethmoidal cell forms the middle wall and the floor of the left optic canal, and the wall of the left third of the optic sulcus. The right sphenoidal sinus forms the wall of the middle third of the optic sulcus.

Figure 67 (Atlas) illustrates this relation, in frontal section. The right sphenoidal sinus here is seen to lie above the nasal septum. This prolongation of the sphenoidal sinus extends from the ostium sphenoidale, 15 millimetres forwards, and comes into relation with the middle third of the optic sulcus and with the chiasma.

30. The left sphenoidal sinus forms the wall of the left third of the optic sulcus. The left posterior ethmoidal cell forms the middle wall and the floor of the optic canal. The right posterior ethmoidal cell forms the middle wall, roof, outer wall, and floor, of the right optic canal, and also the wall of the right third of the optic sulcus.

Group 9.

The sphenoidal sinuses of both sides and the posterior ethmoidal cells of the one side are in relation to the optic nerves.

31. The left sphenoidal sinus forms the middle wall of the left optic canal, and the right sphenoidal sinus forms the middle wall of the right optic canal. The right posterior ethmoidal cell forms the roof of the right optic canal.

Group 10.

There are four varieties of relationship between the optic nerve and the sphenoidal sinus of the one side, and the nerve and the posterior ethmoidal cells of the other side.

32. The left sphenoidal sinus forms the middle wall and floor of the left optic canal, and the whole optic sulcus; it also forms to a

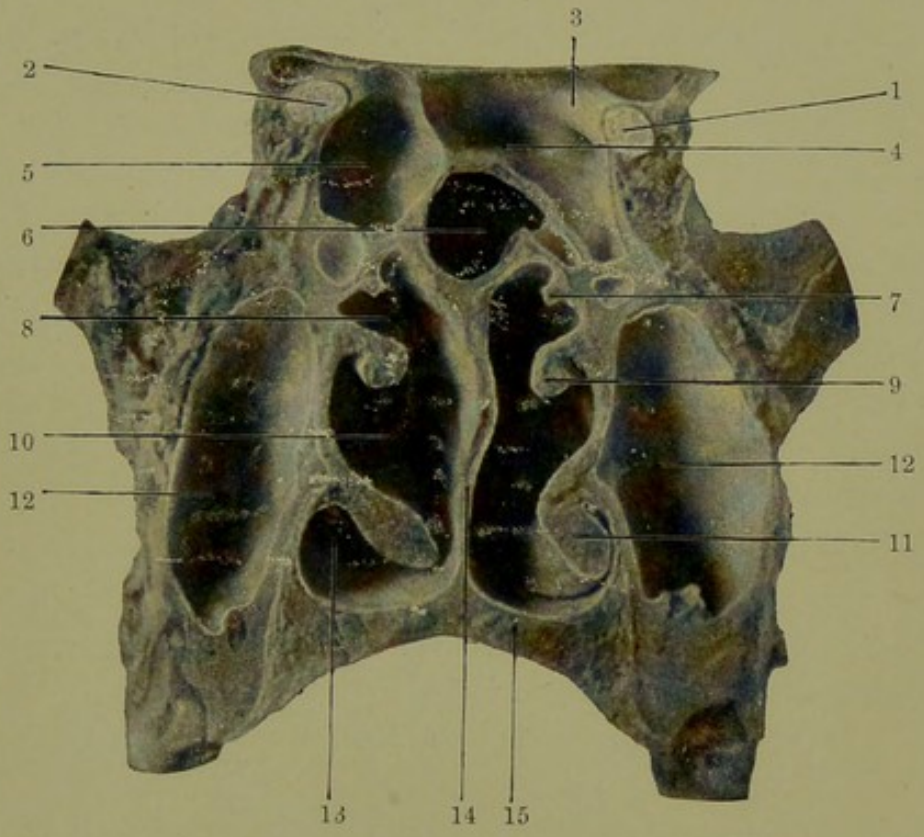


FIG. 32.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3. Optic canal.
4. Left posterior ethmoidal cell.
5. Right sphenoidal sinus.
6. Left sphenoidal sinus.
7. Superior turbinal.
8. Superior meatus.
9. Middle turbinal.
10. Middle meatus.
11. Inferior turbinal.
12. Maxillary antrum.
13. Inferior meatus.
14. Septum.
15. Palate.

small degree the floor and middle wall of the right optic canal, though these are chiefly formed by the right posterior ethmoidal cell.

33. The left most posterior ethmoidal cell forms the middle wall, roof, outer wall and floor, of the left optic canal; the right sphenoidal sinus forms the middle wall, roof, outer wall, and floor of the right optic canal. In Figure 32 and in my Atlas in Figure 72, which illustrates a corresponding frontal section, this relation is shown.

Both figures demonstrate in an instructive manner the relation of the optic nerve and optic canal on the one side to the sphenoidal sinus, and on the other to the posterior ethmoidal cell ; and in addition the course of the optic canal in these cavities is shown for a distance of 12 millimetres. The posterior ethmoidal cell is 28 millimetres long, 30 millimetres broad, and 26 millimetres high. The sphenoidal cavities show a striking asymmetry. On the left side the sinus is 30 millimetres broad, 22 millimetres high, and 41 millimetres long. On the right side it is 20 millimetres broad, 22 millimetres high, and 39 millimetres long. The anterior end of the left sphenoidal sinus lies asymmetrically in the middle line between the lamellæ of the nasal septum.

34. The left sphenoidal sinus forms the middle wall and floor of the left optic canal, and the wall of the left and middle third of the optic sulcus. The right posterior ethmoidal cell forms the middle wall, and to some extent the superior wall, of the right optic canal.

35. The left posterior ethmoidal cell forms the middle wall and floor of the left optic canal, the wall of the whole optic sulcus, and the floor of the right optic canal. The right posterior ethmoidal cell forms the middle wall of the right optic canal.

Group 11.

The frontal sinus of the one side comes into relation with the optic nerve. There are two varieties.

36. The frontal sinus of the one side forms the roof of the optic canal of the same side.

37. The continuous cavity which is formed by coalesced ethmoidal cells and by the frontal sinus, with which the former is continuous, forms the inner wall of the optic canal and the wall of the left third of the optic sulcus. In horizontal section this is illustrated in Figures 97 and 98 in my Atlas.

Group 12.

The cells of the superior turbinated bone of the one side are in relation to the optic nerve.

38. The turbinate bone cells—so-called " bullous cells " of the left superior turbinate—form the floor of the left optic canal and the wall of the left third of the optic sulcus. This relation is shown in frontal section in Figure 33. The cell is here 23 millimetres long, 19 millimetres broad, and 13 millimetres high.

In order to complete this part of the subject, two figures are added which show in an instructive way the relations of the accessory sinuses to the ocular and orbital nerves.

Figure 34 represents a preparation showing the position of the frontal sinus, the anterior and posterior ethmoidal cells, and the sphenoidal sinus, and the relation of these cavities to the anterior and posterior ethmoidal nerves and the optic nerves. It gives a general

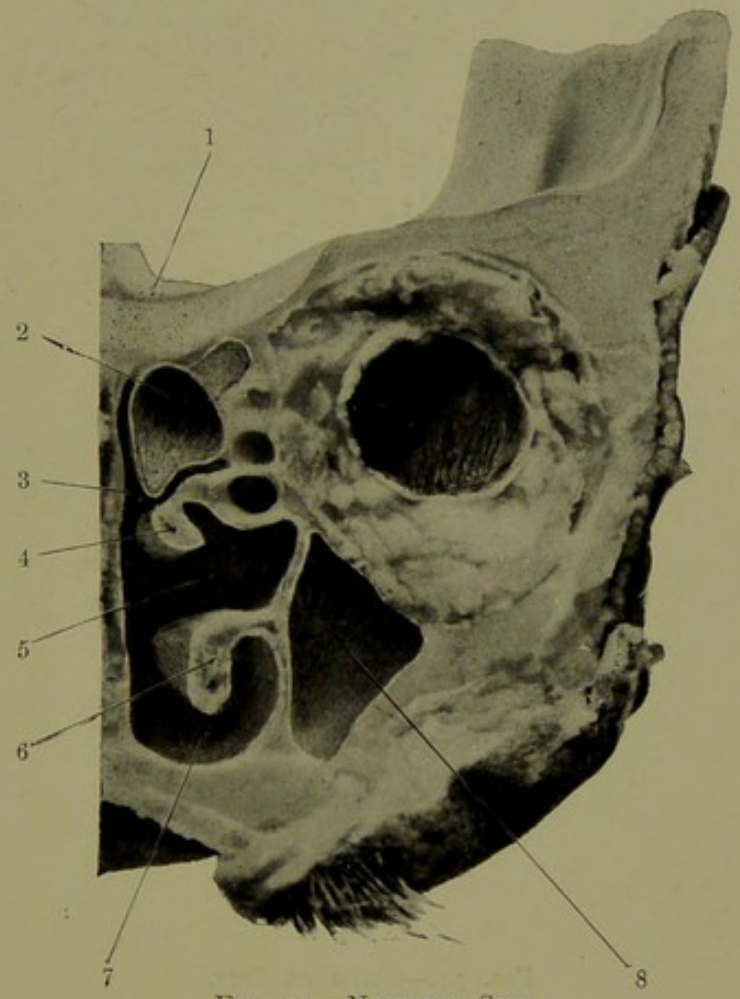


FIG. 33.—NATURAL SIZE.

1. Optic nerve.
2. Cell in the superior turbinal.
3. Superior turbinal.
4. Middle turbinal.
5. Middle meatus.
6. Inferior turbinal
7. Inferior meatus.
8. Maxillary antrum.

view of the position and course of the fourth nerve, the fifth nerve and its three divisions, the third nerve, the sixth nerve, the optic nerves, and the internal carotid artery.

Figure 35 shows in sagittal section the relations of the anterior and posterior ethmoidal nerves, of the optic nerve, and of the third, fourth, and sixth nerves. It also shows the relation of these nerves to the internal carotid artery.

Having described these thirty-eight varieties, classed under twelve groups, with the aid of illustrations, I may now proceed to describe

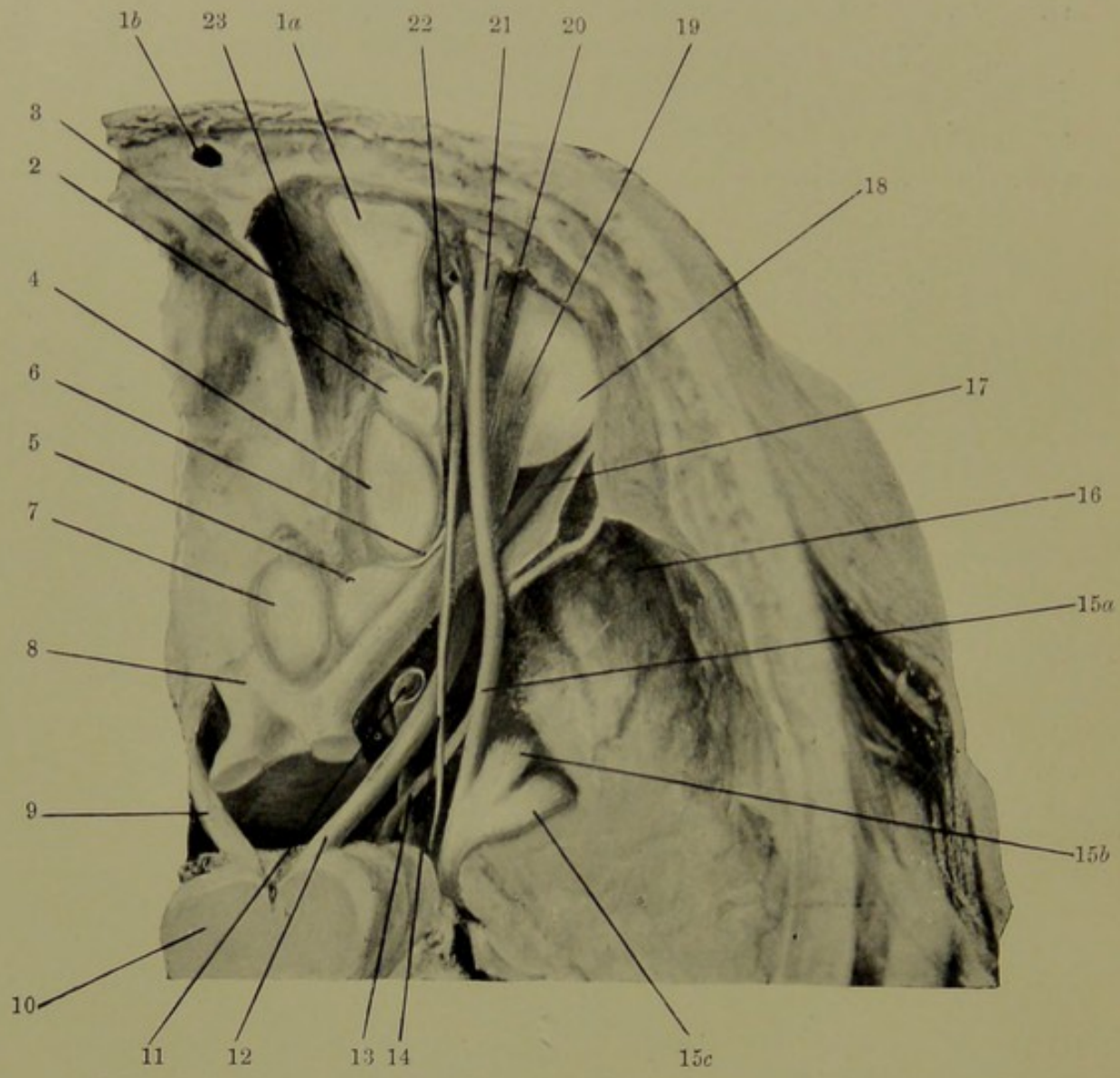


FIG. 34.—NATURAL SIZE.

1*a*. Right frontal sinus.
1*b*. Left frontal sinus.
2. Anterior ethmoidal cell.
3. Anterior ethmoidal nerve.
4. Posterior ethmoidal cell.
5. Right sphenoidal sinus.
6. Posterior ethmoidal nerve.
7. Left sphenoidal sinus.
8. Optic nerves and chiasma.
9. Left third nerve.
10. Pons.
11. Internal carotid artery.
12. Right third nerve.
13. Sixth nerve.
14. Fourth nerve—trochlear.
15*a*, *b*, *c*. Fifth nerve—three divisions.
16. Middle cranial fossa.
17. External rectus.
18. Eyeball.
19. Superior rectus.
20. Levator palpebræ superioris.
21. Supra-orbital nerve.
22. Superior oblique muscle.
23. Anterior cranial fossa.

further such anatomical features as may be considered of practical significance and of ætiological importance. In visual disturbances

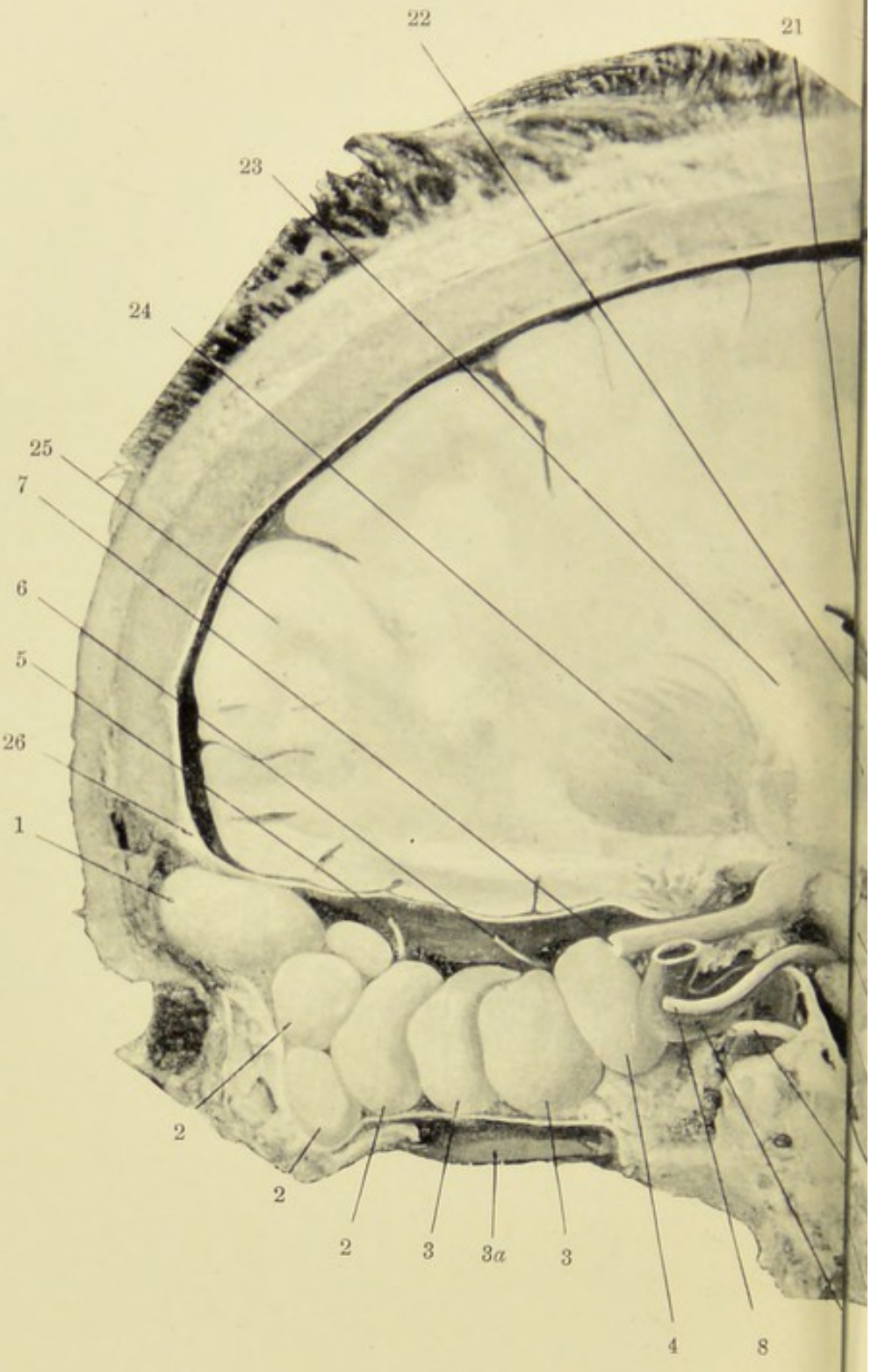
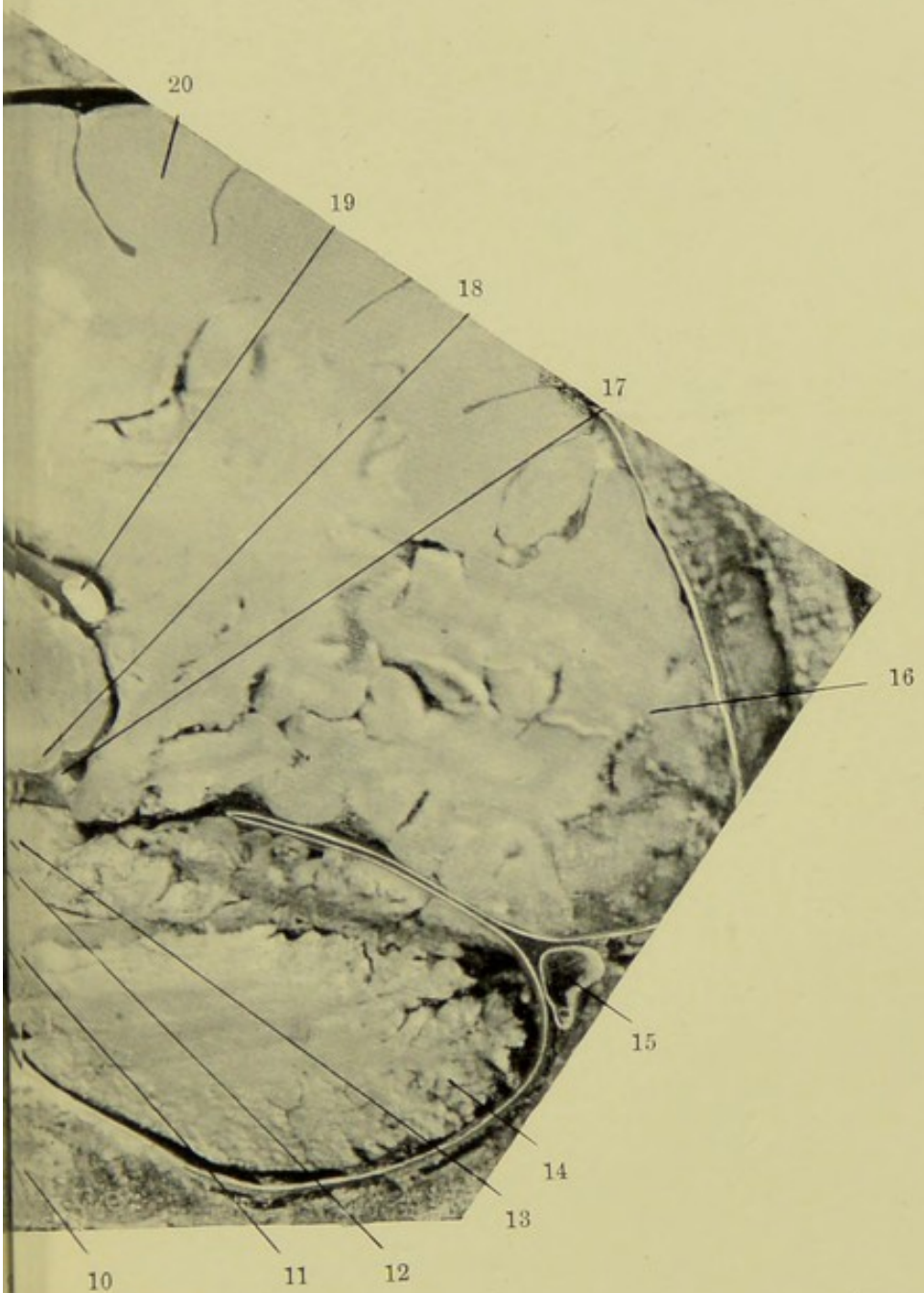


FIG. 35.—

1. Frontal sinus.
2. Anterior ethmoidal cells.
3. Posterior ethmoidal cells.
3*a*. Middle turbinal.
4. Sphenoidal sinus.
5. Anterior ethmoidal nerve.
6. Posterior ethmoidal nerve.
7. Right optic nerve.
8. Internal carotid artery.
9. Left optic
10. Sixth nerv
11. Pons.
12. Fourth ne
13. Cerebral p
14. Cerebellum
15. Straight si
16. Occipital l
17. Crus cereb



TURAL SIZE.

3.
oducens.

trochlearis.
cle.

t corpora quadrigemina.

18. Corpora quadrigemina.
19. Fornix.
20. Parietal lobe.
21. Lateral ventricle.
22. Optic thalamus.
23. Internal capsule.
24. Lenticular nucleus.
25. Frontal lobe.
26. Dura mater.

and blindness caused by disease of the accessory sinuses the following anatomical conditions play an important rôle :

1. The bone wall of the optic canal and of the optic sulcus.
2. Dehiscence in the walls of the sinuses.
3. The semicanalis ethmoidalis.
4. The partition between individual sinuses.
5. The turbinate bone cells.

Each of these will be considered separately.

1. The Bone Wall of the Optic Canal and Optic Sulcus.

The progress of an osteitis, liability to necrosis, complications of the circulatory system, pressure symptoms, and the liability to fracture, may be favoured, made more difficult, or be prevented, according to the thickness of the bone which intervenes between the optic nerve and its neighbouring structures, such as the sphenoidal or ethmoidal sinuses.

Berger and Tyrmann[1] first investigated the degrees of thickness occurring in the bone partition between the optic nerves and the sphenoidal sinuses. In three of their illustrations they represent these facts ; the sections were frontal and in the plane of the optic foramina. In the first illustration this septum is " uncommonly thin "; in the second its thickness reaches 4 to 6 millimetres—in this the bone was spongy ; in the third, on one side the bone was as thin as paper, and on the other 7 millimetres in thickness.

From my own observations, I find that between the most posterior ethmoidal cell and the optic canal and sulcus the bone is in most cases very thin—sometimes as thin as silk - paper. In two cases measured, in the one the thickness was 1 millimetre, and in the other 2 millimetres.

An exceptionally thin partition between the optic nerve and the posterior ethmoidal cell is shown in Figure 15 (Atlas), which represents a sagittal section.

Between the optic canal and sulcus and the sphenoidal sinuses I have more often found a thicker partition. It has varied from 1 millimetre to 5 millimetres, 9 millimetres, and 12 millimetres.

In Figures 17 and 36 wide variations are illustrated. In Figure 17, shown in frontal section, the partition between optic canal

[1] *Loc. cit.*, pages 91-94.

and sphenoidal sinus is thin. But in Figure 36, also in frontal section, is illustrated the thickest partition seen by me.

In Figure 6 the partition between optic nerve and sphenoidal sinus is 6 millimetres thick.

In Figure 37, illustrated in frontal section, the thickness of bone between the chiasma and the sphenoidal sinus is 8 millimetres.

In such cases, where for a varying length the optic canal runs on the internal side of the wall of sphenoidal sinus or ethmoidal cell, its

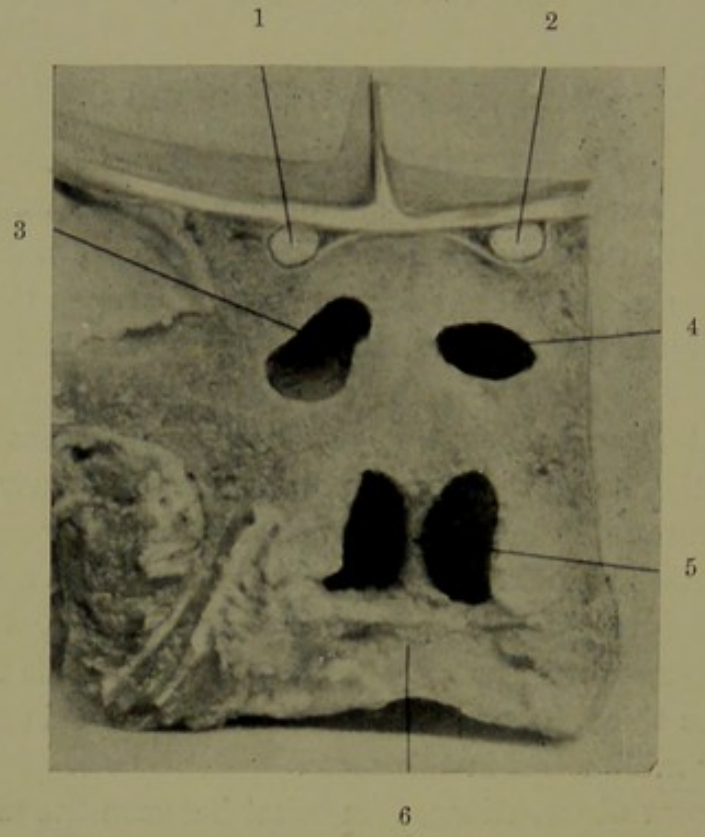


FIG. 36.—NATURAL SIZE.

1. Left optic nerve.
2. Right optic nerve.
3 and 4. Sphenoidal sinuses.
5. Septum.
6. Palate.

wall is generally thin. In Figure 10 an optic canal is represented, which for a distance of 12 millimetres runs within the posterior ethmoidal cell, its upper, inner, and lower walls being bounded by the cell.

Figure 32 and also Figure 72 (Atlas) show the course of optic nerves in the sinuses, and the thinness of their canals.

In Figure 32 is shown, in frontal section, the position of the optic canal in the sphenoidal sinus on the right side, and in the posterior ethmoidal cell on the opposite side. Its length in these cavities

reaches 12 millimetres; its walls on the upper, outer, and inner and lower surfaces are bounded on the right side by the cavity of the sphenoidal sinus, and on the left by that of the posterior ethmoidal cell.

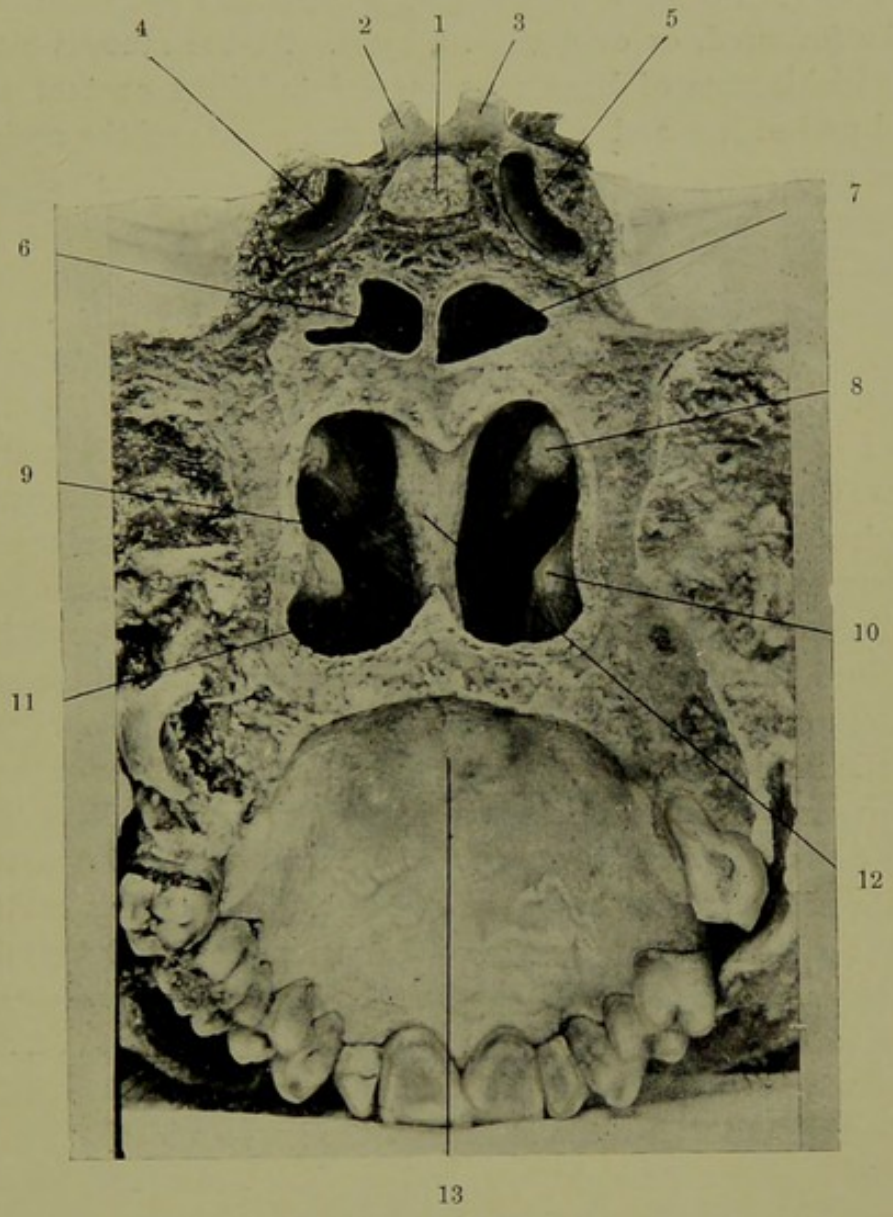


FIG. 37.—NATURAL SIZE.

1. Hypophysis.
2. Left optic tract.
3. Right optic tract.
4. Left internal carotid artery.
5. Right internal carotid artery.
6. Left sphenoidal sinus.
7. Right sphenoidal sinus.
8. Middle turbinal.
9. Middle meatus.
10. Inferior turbinal.
11. Inferior meatus.
12. Septum.
13. Palate.

The position of the optic canal in the posterior ethmoidal cell is shown in Figure 11, in sagittal section. Its length in this is 10 millimetres, and it is surrounded on all sides by the cavity.

2. Dehiscence in the Walls of the Accessory Sinuses.

A dehiscence in a sinus wall is of importance, inasmuch as, in disease of such a sinus, orbital, intracranial, and optic complications may be favoured, or even caused, by it. As the mucous membrane over a dehiscence of bone may come into direct contact with the orbital periosteum and with the dura and sheath of the optic nerve, disease may spread from a sinus cavity to any of these neighbouring structures. In a similar manner the bloodvessels adjacent to a diseased sinus cavity may be affected.

Pathological processes, injury, or senile atrophy may produce an opening in a sinus wall. Such a defect in the bone may be due to an artefact.

As I have already discussed these factors in a previous article,[1] I will here limit myself to the congenital, structural bone defects.

Zuckerkandl[2] describes four cases of bone dehiscence in the maxillary antrum, and Merlin[3] two cases. I have not seen any case. In all Zuckerkandl's cases the defect involved not only the orbital wall of the sinus, but also the lamina papyracea or os planum of the ethmoid, and in one case the tuber maxillæ was involved in addition.

Merlin in his two cases observed the defect in the floor of the orbit.

As regards the frontal sinuses, in the space which lies above the orbital cavity between bone lamellæ, I have observed in parts a few punctate or linear dehiscences placed in the roof of this cavity, and in parts extreme thinning of this bone lamella. Not only openings for bloodvessels, but also actual congenital dehiscence, may occur in the anterior wall of the frontal sinus.

Zuckerkandl and Helly[4] have in one case observed a groove extending upwards from the middle of the supra-orbital ridge, and in this groove areas were defective, and produced communications with the frontal sinus. In a patient I once saw such a congenital defect in the anterior wall of the frontal sinus. Through this a polypus from the sinus protruded.

Helly observed openings for the bloodvessels, the channels of which brought the frontal sinus into communication with the anterior surface

[1] Ónodi, "Dehiscence in the Walls of the Nasal Accessory Sinuses," *Archives für Laryngologie*, Bd. XV.

[2] "Anatomy of the Nasal Cavities," 1893.

[3] Report of the Naturwissenschaftlich-Medizinischen Vereines in Innsbruck, 1884-85, 1885-86.

[4] *Deutsche Zeitschrift für Chirurgie*, Bd. III.

of the frontal bone. Such channels of smaller and larger size I have observed in the supra-orbital ridge. Those cases of congenital dehiscence in which the floor of the frontal sinus and the lamina papyracea or os planum of the ethmoid are involved in common are of rare occurrence. Zuckerkandl found this in three cases—twice on the left and once on the right side. Merlin saw it once on the left side.

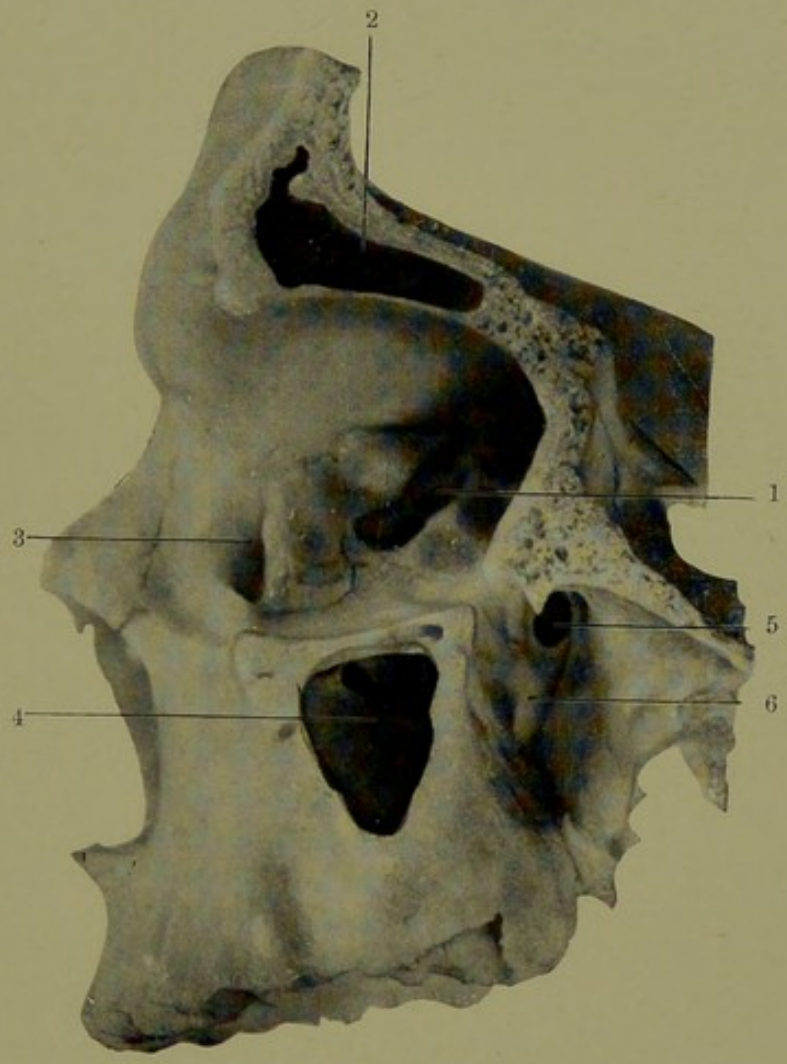


FIG. 38.—NATURAL SIZE.

1. Dehiscence.
2. Frontal sinus.
3. Lachrymal groove.
4. Maxillary antrum.
5. Spheno-palatine foramen.
6. Pterygo-palatine fossa.

I have seen three such cases—twice on the right and once on the left side.

According to the observations of Denouvilliers and Gosselin,[1] Winkler,[2] and Killian,[3] small or large openings may exist in the inter-

[1] "Compend. de Chir. Prat.," 1852. [2] *Archiv für Laryngologie*, 1894.
[3] *Münchener Medizinische Wochenschrift*, 1897.

frontal septum, thus placing the two frontal sinuses in communication. Such an opening, measuring 1·5 millimetres in diameter, I observed once. Its edges were smooth and rounded.

Forty years ago Hyrtl[1] described dehiscence in the lamina papyracea of the ethmoid ; more recently Zuckerkandl observed it in fourteen cases, nine being on the left side. Merlin saw it three times —twice on the left and once on the right side.

In one case five dehiscences were present in this ethmoidal plate, and through these the orbital cavity, and the frontal and sphenoidal sinuses were placed in communication with the ethmoidal cells.

I have found eighteen cases of dehiscence of the lamina papyracea—thirteen on the left and five on the right side. The sagittal measurement of these defects varied between 4 and 28 millimetres, and the vertical between 4 and 8 millimetres. In this manner the orbital cavity communicated with the ethmoidal cells, and in three cases also with the frontal sinus. Figure 38 shows a specimen which illustrates a dehiscence in the ethmoidal orbital plate.

Dehiscences of physiological significance have been observed by Zuckerkandl in the form of small fissures in the side-wall of the sphenoidal sinus. These place the sinuses in communication with the middle fossa of the skull.

Spec[2] observed a defect in the bone wall of the carotid canal.

In several skulls I have observed apertures for bloodvessels, often symmetrically placed, immediately under the side-wall of the base of the small wing of the sphenoid. In a few cases, leading to these vascular apertures are vascular grooves, in which more or less lengthy linear dehiscences are apparent.

In the intersphenoidal septum, Zuckerkandl and Hajek have noted dehiscence, placing the two sphenoidal sinuses in communication.

The wall of the optic canal rarely shows dehiscence. It was found twice in 200 cases by Gallmaerts[3] ; in 50 cases Holmes[4] found it twice ; in 300 cases I found it once.

3. Semicanalis Ethmoidalis.

A canal of varying length, and termed by me the "semicanalis ethmoidalis,"[5] runs in the wall of the frontal sinus or in the orbital

[1] *Vergangenheit und Gegenwart des Museums für Menschliche Anatomie*, 1869.
[2] *Bardelebens Handbuch der Anatomie*, 1896.
[3] *Annal. d'Ocul*, 1900.
[4] *Arch. of Ophthalm.*, 1896.
[5] Onodi, *Archiv für Laryngologie*, Bd. XIV and XV.

cells to the anterior fossa of the skull. In it, through these cavities and freely covered with mucous membrane, pass the ethmoidal veins. The mucous membrane of this open canal at one end touches the orbital periosteum and at the other the dura mater.

Owing to the connections of the ethmoidal veins with the venous plexuses both of the dura mater and of the orbital cavity, a thrombophlebitis may spread in either direction, as Zuckerkandl and Kuhnt have pointed out. The ethmoidal veins of the semicanalis ethmoidalis may in a similar manner produce thrombosis of the cerebral and orbital veins.

Generally a very short canal—the canalis ethmoidalis—is formed out of the apposed grooves in the frontal and ethmoidal bones. It commences in the anterior ethmoidal foramen, and ends in the

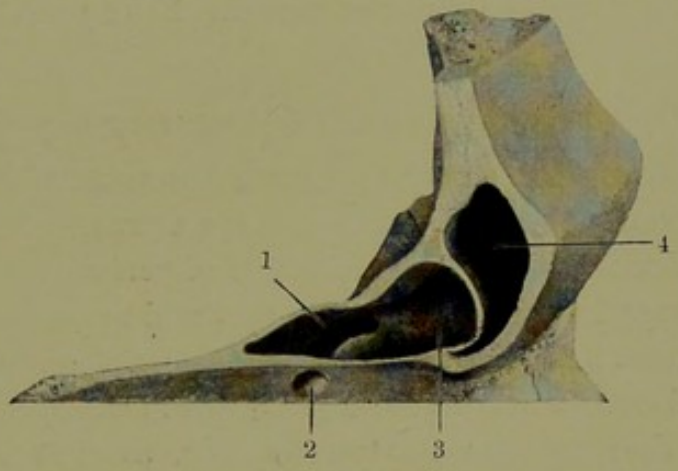


FIG. 39.—NATURAL SIZE.

1. Semicanalis ethmoidalis.
2. Anterior ethmoidal foramen.
3. Fronto-orbital cell.
4. Frontal sinus.

anterior fossa of the skull as a linear groove. According to my observations, the length of the canalis ethmoidalis varies between 5 and 12 millimetres.

Such ethmoidal cells as extend into the horizontal part of the frontal bone I term the "orbital cells." There may be one or two orbital cells that open into the middle or superior meatus of the nose.

Figure 39 illustrates in sagittal section the course of the semicanalis ethmoidalis in such an orbital cell. The whole course of this open canal is shown, lying in a large orbital cell, and connecting through this cavity the anterior fossa of the skull internally with the orbital cavity externally. The anterior ethmoidal foramen is continued into this ethmoidal open canal, which for a distance of 10 milli-

metres passes in a crescentic course on the posterior and inner wall of the orbital cells, to end in the anterior cranial fossa.

Three times have I observed the semicanalis ethmoidalis in the frontal sinus, with a length varying between 5 and 8 millimetres. Nine times did it manifest itself in the first orbital cell, with a length varying between 7 and 10 millimetres, and in the second orbital cell four times, with a length of between 4 and 10 millimetres. Twice in association with a dehiscence in the lamina papyracea did I see a semicanalis ethmoidalis in the orbital cells; and once, as is illustrated in Figure 97 (Atlas) a semicanalis 16 millimetres in length was found passing downwards on the lateral wall of the frontal sinus. In this skull the cavity in the ethmoid was continuous with the frontal sinus, and the distance between the anterior wall of the frontal sinus and the anterior ethmoidal foramen amounted to 23 millimetres.

4. Septa between Individual Accessory Sinuses.

I will now discuss such very thin septa between individual sinuses as I have personally observed, for these have a practical importance in the spread of disease and in the production of perforations. The maxillary antrum may have a wall common to it and to the sphenoidal and ethmoidal sinuses. And, as I was the first to point out, the maxillary and sphenoidal sinuses may be so large as to lie in direct apposition.[1]

Figure 40 demonstrates this relationship, as seen in horizontal section. The maxillary antrum was 42 millimetres long and 36 millimetres broad; the sphenoidal sinus 37 millimetres high, 47 millimetres broad, and 35 millimetres long. The width of septum common to these cavities was 10 millimetres, and it formed at the same time the maxillary part of the anterior sphenoidal wall. Such partitions common to these two cavities I have observed in six cases.

The width of this partition—that is, the maxillary part of the anterior wall of the sphenoidal sinus—was in two cases 4 millimetres, in one 6 millimetres, in another 8 millimetres, and in two other cases 10 millimetres.

Thin septa are also present between the maxillary antrum and the ethmoidal cells. Such a partition common to the maxillary antrum and the posterior ethmoidal cells, present on both sides, is illustrated in Figure 41.

The extreme thinness of such a septum is illustrated in Figure 42

[1] *Archiv für Laryngologie*, Bd. XI.

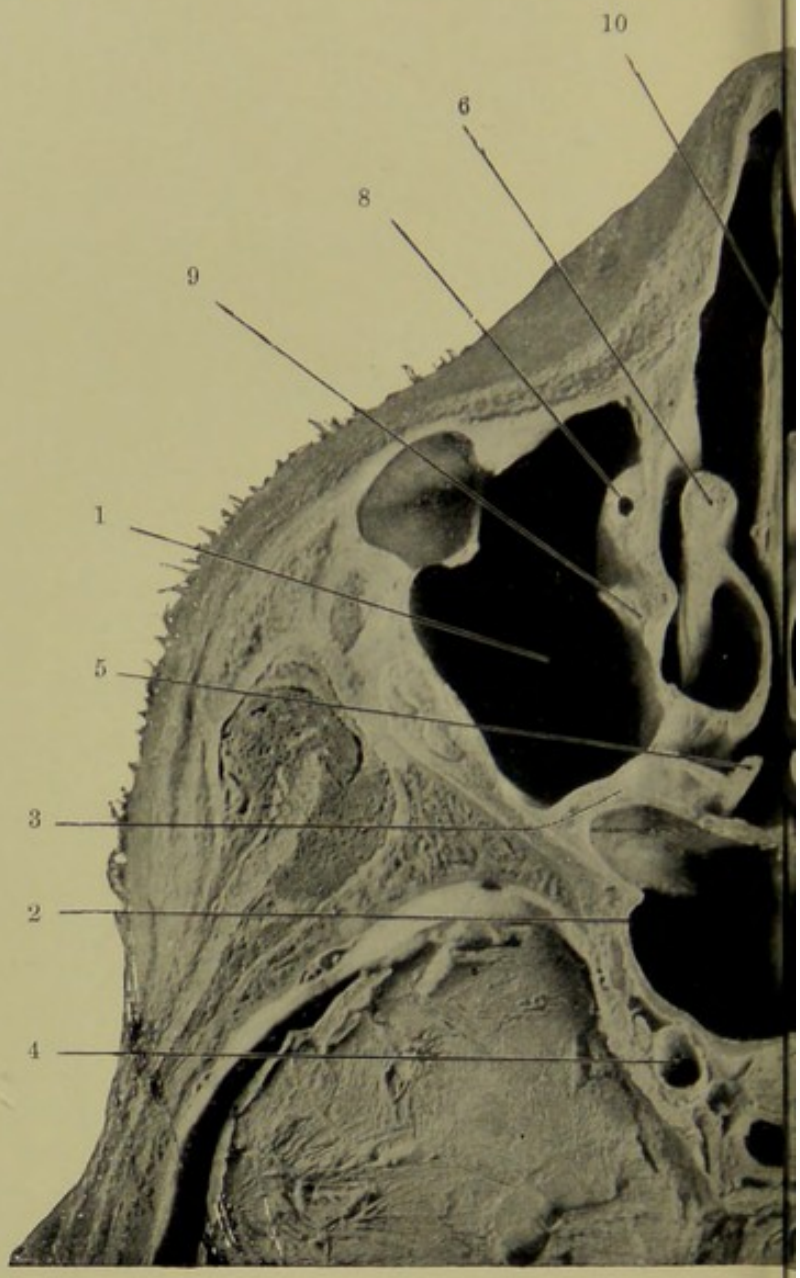
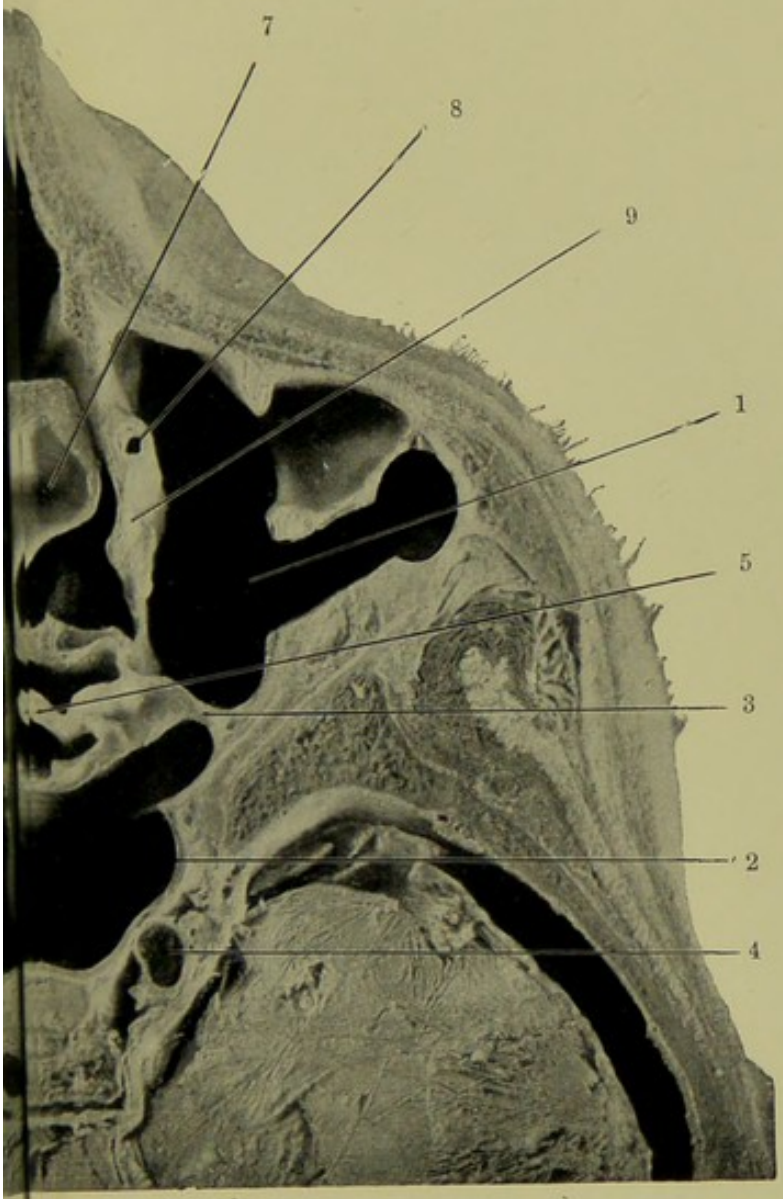


FIG. 40.—N

1. Maxillary antrum.
2. Sphenoidal sinus.
3. Pars maxillaris antri.
4. Internal carotid artery.
5. Superior turbinal.



ERAL SIZE.

. Middle turbinal.
. Cell in middle turbinal.
. Naso-lachrymal duct.
. Inner wall of maxillary antrum.
. Septum.

in frontal section, and in Figure 48 its presence on both sides is shown, the section being horizontal.

The frontal sinus may have a septum common to it and to the sphenoidal sinus and the posterior ethmoidal cells. The septum that intervenes between the two frontal sinuses may lie sagittally, or, where there is asymmetry, may assume an oblique position.

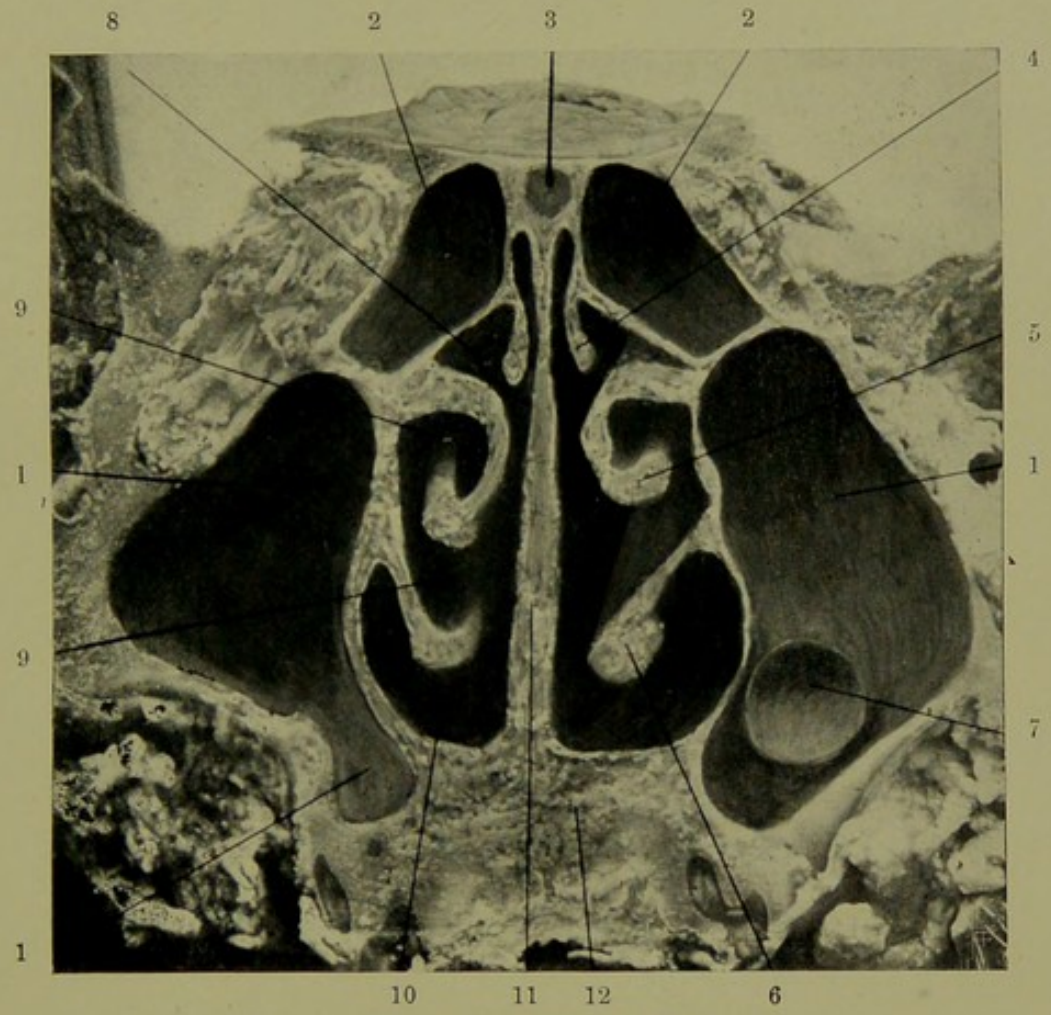


FIG. 41.—NATURAL SIZE.

1. Maxillary antrum.
2. Posterior ethmoidal cell.
3. Left sphenoidal sinus.
4. Superior turbinal.
5. Middle turbinal.
6. Inferior turbinal.
7. Cyst.
8. Superior meatus.
9. Middle meatus.
10. Inferior meatus.
11. Septum.
12. Palate.

In Figure 103 (Atlas) a sagittal septum medially placed is shown. In Figure 43 an asymmetry of the frontal sinuses is shown in sagittal section. In this the left sinus covers the right over a breadth of 15 millimetres, and upwards it extends yet farther for 30 millimetres. In addition there is a horizontal septum common to these two sinuses besides the vertical one. There may be only one

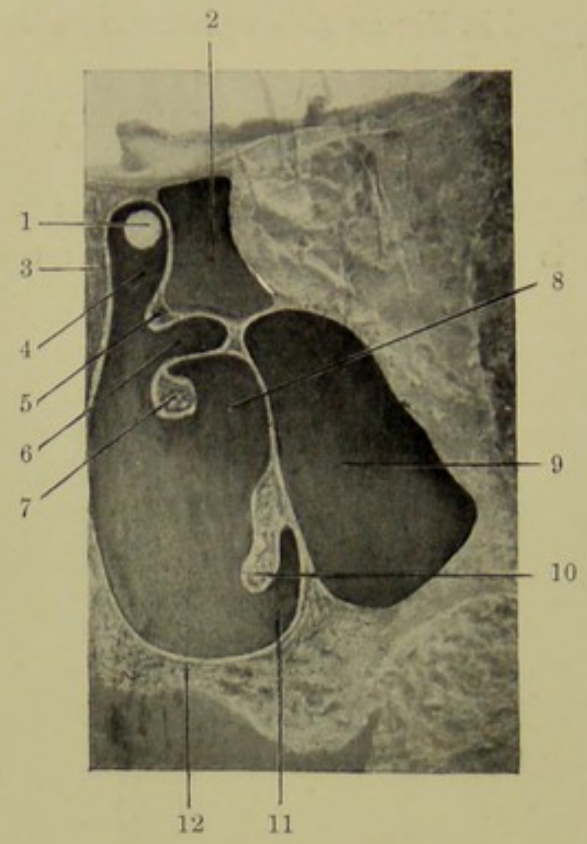


FIG. 42.—NATURAL SIZE.

1. Opening of sphenoidal sinus.
2. Posterior ethmoidal cell.
3. Septum.
4. Nasal part of sphenoidal sinus wall.
5. Superior turbinal.
6. Superior meatus.
7. Middle turbinal.
8. Middle meatus.
9. Maxillary antrum.
10. Inferior turbinal.
11. Inferior meatus.
12. Palate.

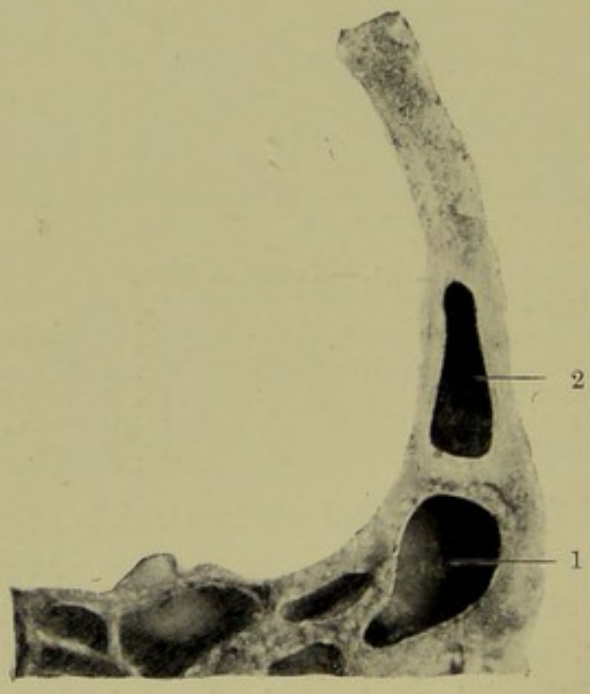


FIG. 43.—NATURAL SIZE.

1. Right frontal sinus.
2. Left frontal sinus.

frontal sinus, or even both may be absent. In such cases an inter-frontal septum is non-existent. Figure 10 (Atlas) illustrates in sagittal section a septum common to the anterior ethmoidal cells and the frontal sinuses. Figures 44 and 45 illustrate a septum common to those ethmoidal cells that stretch to the region of the frontal sinuses—the bulla frontalis and the frontal sinus. In Figure 44 there is well seen in sagittal section the thin septum between the bulla frontalis, which is 17 millimetres long, 22 millimetres high, and 21 millimetres broad; and the frontal sinus, which is 25 millimetres long, 6 millimetres high, and 34 millimetres broad.

In Figure 45 there is shown, in frontal section, on both sides the septum common to the bulla frontalis and the frontal sinus. On the right side the frontal sinus is 20 millimetres long, 30 millimetres broad, and

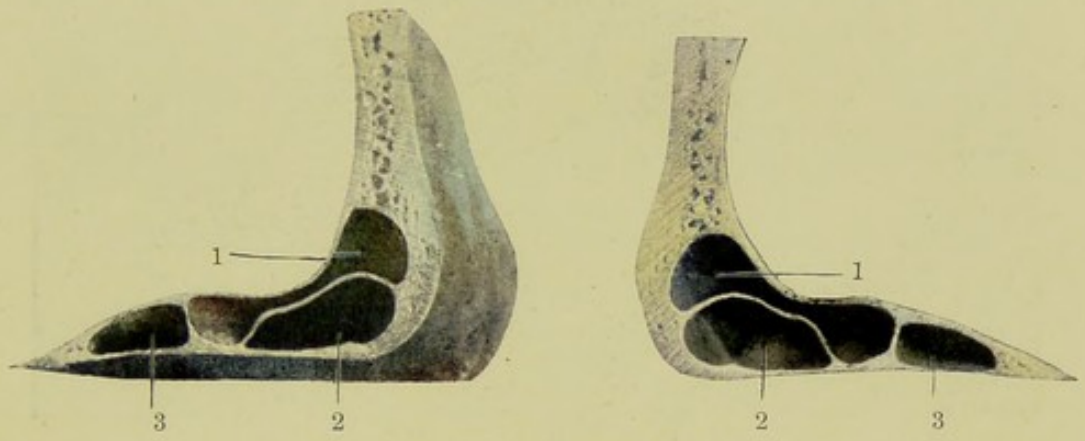


FIG. 44.—NATURAL SIZE.

1. Frontal sinus. 2. Frontal bulla.
3. Fronto-orbital cell.

11 millimetres high, and on the left 14 millimetres long, 30 millimetres broad, and 11 millimetres high. On the right side the bulla frontalis is 20 millimetres long, 13 millimetres broad, and 20 millimetres high, and on the left side 20 millimetres long, 13 millimetres broad, and 20 millimetres high. That is identically the same as on the right side.

The following sagittal sections illustrate a septum common to the frontal sinus and an orbital cell. Figure 46 shows a thin arched septum between the frontal sinus, which is 23 millimetres high, 30 millimetres broad, and 13 millimetres long, and an orbital cell 27 millimetres long, 19 millimetres high, and 29 millimetres broad.

Figure 36 (Atlas) shows a communication between the frontal sinus and the orbital cell.

FIG. 45.—NATURAL SIZE.

1. Frontal sinus.
2. Frontal bulla.
3. Partition between frontal sinus and frontal bulla.
4. Eyeball.
5. Duct.
6. Middle turbinal.
7. Maxillary antrum.
8. Inferior turbinal.
9. Inferior meatus.
10. Superior turbinal.
11. Opening of maxillary antrum.
12. Septum.
13. Palate.

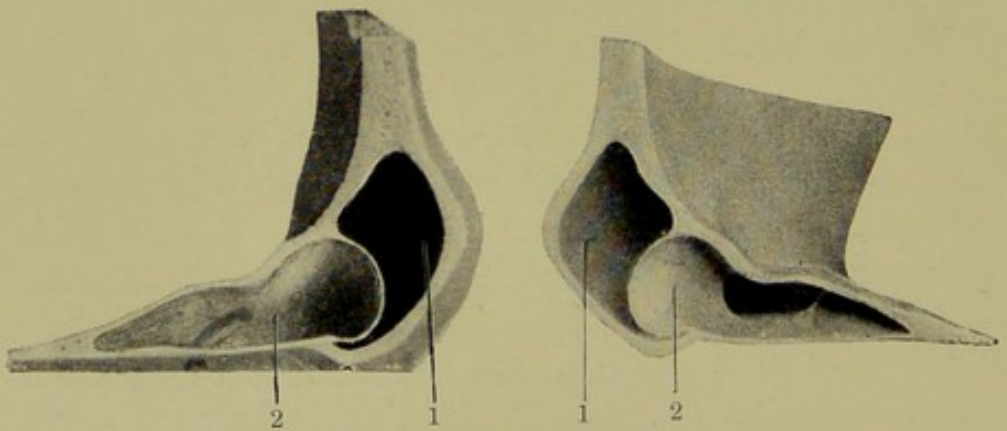


FIG. 46.—NATURAL SIZE.

1. Frontal sinus.
2. Fronto-orbital cell.

Figure 47 shows an exceptionally thin septum common to the frontal sinus which is 19 millimetres long, 35 millimetres high, and 32 millimetres broad, and an orbital cell which is 25 millimetres long, 28 millimetres broad, and 13 millimetres high.

Figure 26 (Atlas) illustrates a case in which the frontal sinus is separated from the posterior ethmoidal cell by a septum common to the two cavities; and Figure 8 shows a specimen in which the ethmoidal cells form a continuous cavity with the frontal sinus.

In Figures 31, 37, 52 (Atlas), are illustrated in sagittal section the septa common between the frontal sinuses and both orbital cells, and also the communications between individual cavities. Figure 31 shows a thin septum between the frontal sinus and the first orbital cell, and a thin septum between the first and second orbital cells. In Figure 37 is shown a complete septum between

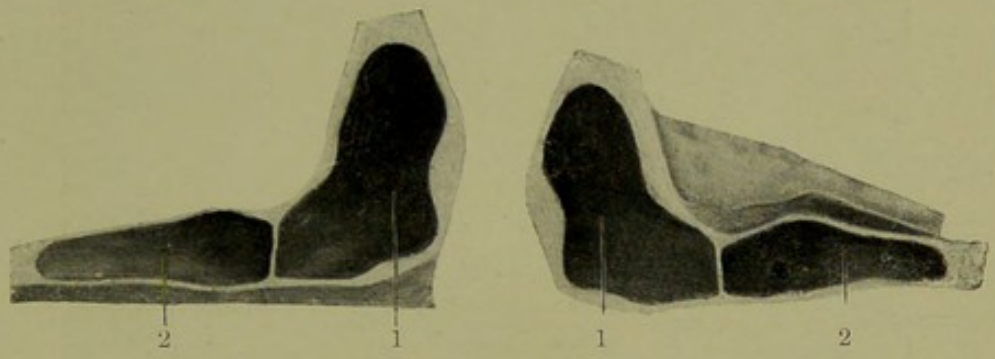


FIG. 47.—NATURAL SIZE.

1. Frontal sinus. 2. Fronto-orbital cell.

the first and second orbital cells, and a communication between the first orbital cell and the frontal sinus. In Figure 52 a complete septum separates the frontal sinus and the first orbital cell, and there is a communication between the first and second orbital cells. In Figure 38 (Atlas) a communication is shown between the frontal sinus and the two orbital cells. The frontal sinuses may extend far back, even up to the level of the optic foramina, and in such cases they may be separated from the posterior ethmoidal cell as well as from the sphenoidal sinus by quite a thin septum.

Figure 65 (Atlas) illustrates a preparation in which the frontal sinuses extend far back over the orbital roofs, and on the right side the frontal sinus is only separated from the right sphenoidal sinus by a thin septum, and on the left side from the left posterior ethmoidal cell by a thin septum. This septum between the sphenoidal sinus and the frontal sinus—in other words, the pars frontalis of the

anterior wall of the sphenoidal sinus—measures 12 millimetres in breadth.

In Figure 66 (Atlas) a specimen is illustrated in frontal section in which the frontal sinus extends back to the region of the sphenoidal sinus and of the optic nerve, and is separated from the posterior ethmoidal cell by a thin partition.

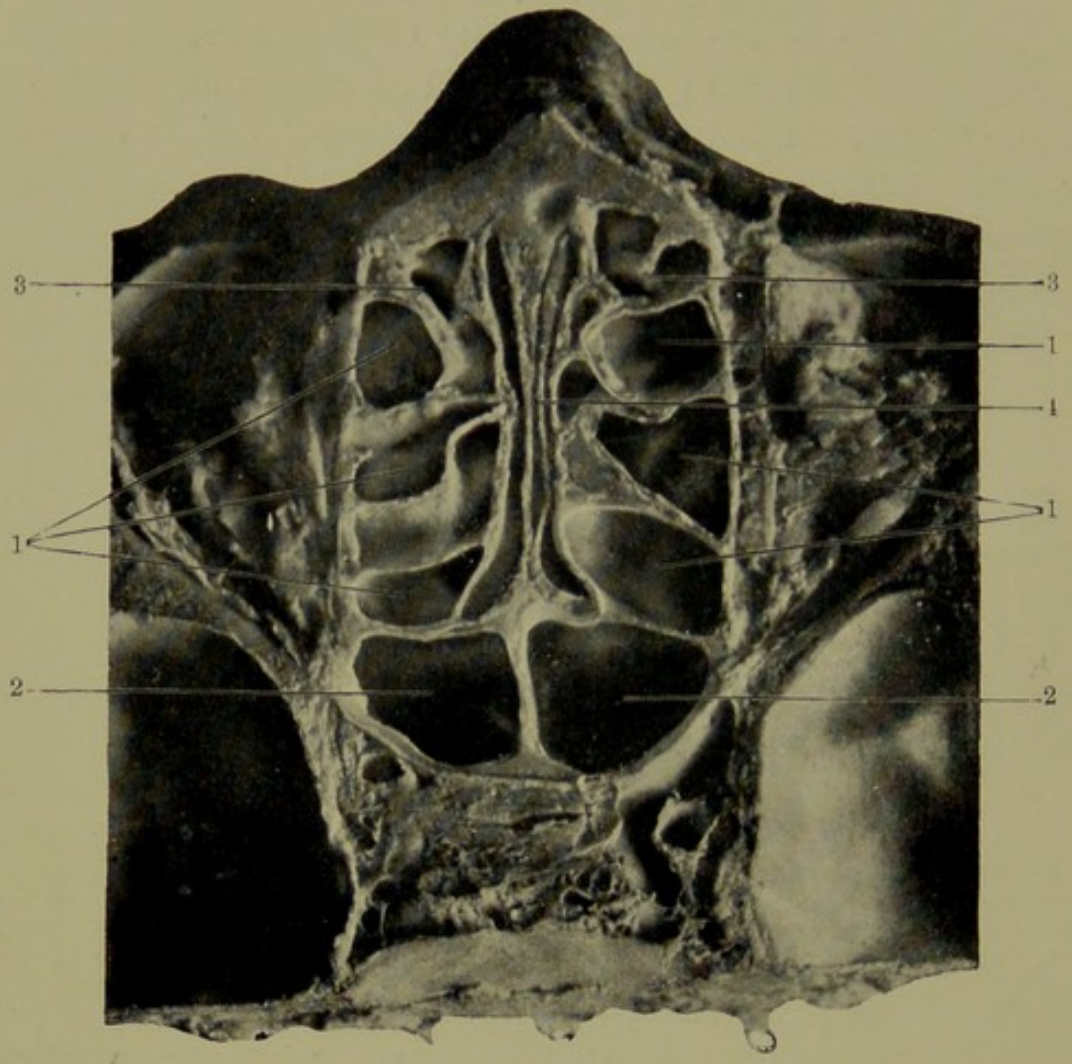


FIG. 48.—NATURAL SIZE.

1. Ethmoidal cells.
2. Sphenoidal sinus.
3. Naso-frontal duct.
4. Septum.

In Figures 48 and 49 the septa between individual ethmoidal cells are shown.

Finally, there remain to be considered the septa between the posterior ethmoidal cells and the sphenoidal sinuses. The posterior ethmoidal cells may lie in series above the sphenoidal sinus, or may lie in front of it. The septum intervening may be vertical or horizontal or oblique. This area of the sphenoidal wall, the pars ethmoidalis

of that wall, may vary in width between 1 millimetre and 18 millimetres. In Figure 49 the septum between the posterior ethmoidal cell and the sphenoidal sinuses, as well as the intersphenoidal septum, are shown in horizontal section. In this specimen the intersphenoidal septum lies in a sagittal plane, but in the presence of asymmetry of

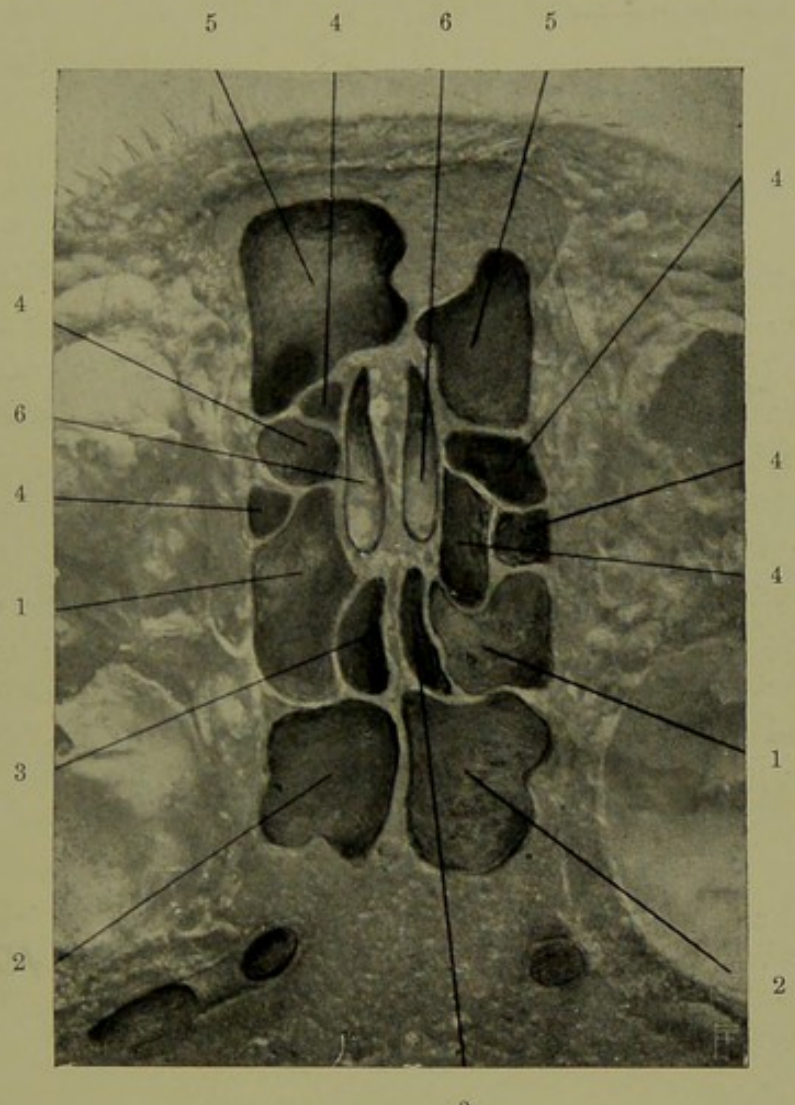


FIG. 49.—NATURAL SIZE.

1, 1. Posterior ethmoidal cells.
2, 2. Sphenoidal sinuses.
3, 3. Olfactory fissures.
4, 4, 4, 4 4. Anterior ethmoidal cells.
5. Frontal sinus.
6. Surface of anterior cranial fossa.

the sinuses it may lie in various planes, and may have bendings, or, in the absence of one sinus, may be non-existent.

In Figure 41 a rare relationship is shown in frontal section. The right sphenoidal sinus has a septum common between it and both posterior ethmoidal cells. In Figure 32 a similar relation is shown between the left sphenoidal sinus and both posterior ethmoidal cells.

Figure 15 shows in sagittal section the most posterior ethmoidal cell, placed above the sphenoidal sinus, and an exceptionally thin septum between the two cavities.

5. Turbinate Bone Cells, or Osseous Bullæ.

The osseous bullæ, which I term the "turbinate bone cells," are a developmental anomaly.[1] Owing to disease, they may reach a very varying size. They may be present in both superior and middle turbinated bones. Those of the superior turbinated bone may come into relation with the optic nerves. As they may produce pressure symptoms due to the enlargement and distension caused by pathological changes, their clinical importance is considerable. I have seen such cells nine times in the middle turbinate and twice in the superior turbinate. Only in two cases did they open into the middle meatus; in other cases they opened into the superior meatus. In length they varied from 8 to 20 millimetres, in breadth from 5 to 14 millimetres, in height from 7 to 16 millimetres.

In Figure 18 (Atlas) a cell in the middle turbinate is shown in sagittal section; it is 17 millimetres long, 20 millimetres high, and 14 millimetres broad. In Figure 33, in frontal section, a cell is shown in the upper turbinated bone; it is 23 millimetres long, 19 millimetres broad, and 13 millimetres high. This cell reaches posteriorly up to the region of the optic nerves.

[1] Ónodi, *Archiv für Laryngologie*, Bd. XV.

II

CLINICAL CONSIDERATIONS

Our knowledge of the production of visual disturbance by accessory sinus disease is, unfortunately, still defective.

Traumatic lesions are shown, through post-mortem examination, to be produced by swellings arising from the naso-pharynx, from the sphenoidal sinuses, the ethmoidal cells, or from the ethmoid bone itself. Either by pressure or through circulatory interference the optic nerves become impaired in function. Post-mortem examinations also reveal traumatic lesions of the optic nerve caused by fracture or fissuring in the region of the sphenoid, or the involvement of the nerve in a hyperostosis of the wing of the sphenoid causing narrowing of the optic foramen. Post-mortem examination further reveals the association of accessory sinus suppuration with perforation of sinus walls, caries and necrosis, basal meningitis, extra- and intradural abscess, and thromboses of the cavernous sinus and ophthalmic veins. There are only a few observations, both microscopic and bacteriological, which prove the source and spread of the infection by continuity and by the bloodvessels. In the post-mortem examination there remain to be demonstrated the manner and course of the spread of the disease, with causal relationships; also microscopic and bacteriological investigation of the diseased sinuses and of the optic nerves. What is also lacking is the examination of the ethmoidal veins, the central vein of the retina, and the venous radicles arising from the diseased sinuses; and we also need a description of the exact relations of the optic nerves to the diseased and healthy sinuses.

To the isolated pathological facts at our disposal we can add observations made clinically and draw deductions from these. Such observations concern the clinical bearings of visual disturbances and blindness upon disease of intranasal origin, and the production of optic neuritis and atrophy in the optic canal. To preface a consideration of these clinical symptoms, I may add that various causes

have been established by ophthalmologists as productive of optic neuritis and optic atrophy. Optic neuritis or optic atrophy may be present in disease of the nasal accessory sinuses without having been caused by it. Even in cases where the nasal origin of such optic nerve disturbance can no longer be doubted, we must still bear in mind the possibility of accidental association.

Cases have been observed where the unfavourable progress of an optic neuritis was not in the slightest degree influenced by operation on diseased accessory sinuses ; and, on the other hand, in cases of accessory sinus suppuration, in which operation was refused, spontaneous cure of an optic neuritis that was present has been reported. Cases, too, are not uncommon in which optic neuritis or optic atrophy is in existence, where the ophthalmologist can discover no cause and the rhinologist finds normal nasal cavities. It is seen thus that accessory sinus suppuration may be present in cases of optic neuritis or optic atrophy, and be apparently in causal association, and yet be independent and unassociated with it.

The clinical pictures of these cases of true and apparent causal association of accessory sinus with optic nerve disease so merge into each other that a true diagnosis may be very difficult, or even impossible, clinically. I mention this, as it is my conviction that false inferences are not infrequently made in these cases.

We now pass on to the ætiology of such visual disturbances.

Mechanical and traumatic lesions have been proved to be responsible, by post-mortem examinations. Swellings or tumours in the region of the optic nerves, and arising from the accessory sinuses, may cause symptoms through direct pressure. Bering and Wicherkiewicz[1] have shown a case in which a normal optic nerve lay embedded in a growth. Ponfick, also Schmidt-Rimpler,[2] and Ónodi,[3] have shown that in sarcoma of the body of the sphenoid the optic nerves may remain intact. Virchow[4] and Manz[5] have shown in post-mortem dissections that in early youth developmental anomalies, premature ossification, and hyperostosis in the base of the skull, may cause blindness, in some cases through constriction of the optic nerve at the optic foramen.

Enslin[6] and Goldzieher[7] have observed blindness in cases of

[1] *Berliner Klinische Wochenschrift*, 1882.
[2] *Archiv für Laryngologie*, Bd. XVII.
[3] *Ibid.*
[4] "Krankhafte Geschwülste."
[5] *Heidelberger Berichte*, 1887.
[6] Graefes, *Archiv für Ophthalmologie*, Bd. LVIII.
[7] *Budapesti Orvosi ujság Szemészet*, 1904.

dolichocephalics. In two cases I have seen anosmia associated with blindness, due to constriction of the foramina of the olfactory and optic nerves.

Direct and indirect fracture of the optic canal and injury to the optic nerves is known. Hölder[1] reports fifty-three cases of this nature. In all of them the ætiology was established by post-mortem examination.

In dealing with *accessory sinus suppuration* as a cause of visual disturbance, we have to consider the nature and spread of the infection, and the complications due to infection of the bloodvessels, as well as the presence of any bone dehiscence. From post-mortem examinations, bone necrosis, thrombo-phlebitis, meningitis, orbital cellulitis, cerebral abscess, etc., are found associated with accessory sinus suppuration. Such findings are, however, as yet incomplete. The exact path of the infective disease, the changes in the walls of the accessory sinuses and in the bloodvessels, especially in the ethmoidal veins and central vein of the retina, and microscopic examination of the optic nerves—these still remain to be investigated. The paths of infection in two cases, through direct continuity and through bloodvessels, have been carefully determined by Ortmann[2] and Hajek.[3]

In Ortmann's case an empyema of the sphenoidal sinus was associated with an extradural abscess in the sella turcica, and thrombosis of both cavernous sinuses. The bone of the sella turcica was of a brownish-red colour. The following observation was made on microscopic examination: "It could be determined that numerous diplococci lying between leucocytic infiltrations had invaded both the mucous membrane and periosteum of the sphenoidal cavity. These showed numerous small hæmorrhages as well. Microscopic sections of the sphenoidal body and its membranes also revealed these diplococci."

Thus the spread of the disease was by direct continuity to the meninges. The inflammation in the sphenoidal sinus, and the damming up of its secretion, led to periostitis and purulent osteomyelitis, which infected both dura and pia mater.

In Hajek's case ethmoidal suppuration was associated with meningitis. On post-mortem examination, a diffuse acute pachymeningitis interna and leptomeningitis with a fibrinous purulent exudate was found both over the base and convexity of the brain. Acute hydro-

[1] Berlin, "Graefe-Sämisch' Handbuch." [2] *Virchow's Archiv*, Bd. CXX.
[3] *Archiv für Laryngologie*, Bd. XVIII.

cephalus of the ventricles was present. The ethmoidal cells on the left side were inflamed, and the maxillary antra showed œdema of their mucous membrane. The intracranial exudate revealed *Streptococcus pyogenes*. Hajek, after a microscopic investigation, concludes as follows with reference to the path of infection : " We had to deal with infiltration of the inflamed mucous membrane tissue by virulent streptococci, and, further, with an invasion of the bloodvessels by the streptococci. On account of the well-known venous anastomoses between the mucous membrane of the ethmoidal cells and the dura mater, without there being any involvement of bone or bone marrow, the meninges became rapidly infected." Westenhöfer's[1] previous statements that a cerebro-spinal meningitis may arise through the lymphatic channels have not been proved by microscopic examination, and he himself in a recent statement[2] says that the lymph channels must yet be proved by microscopic research to be the paths of infection. The material clinically observed by me, and which came to the post-mortem room, as well as other collected material, are being examined as to their pathology by Dr. B. Entz, assistant to the Pathological Anatomical Institute at Budapest. These researches are as yet incomplete.

Such observations, clinical and pathological, as concern complications possible to individual diseased accessory sinuses will now first be reviewed, and later the ætiology will be more closely considered. These observations will be shortly discussed and taken in groups.

Foucher[3] points out as complications of disease of the *maxillary antra* orbital phlegmon and thrombo-phlebitis of the pterygoid and ophthalmic venous plexuses. Mair[4] describes a case with caries of the ethmoid, perforation of the lamina cribrosa, and abscess in the prefrontal convolutions of the cerebrum ; and Panas[5] found a case with necrosis of a part of the orbital roof, and purulent periostitis and abscess in the frontal convolutions ; and Dmochovszky[6] found several perforations in the walls of the maxillary antrum, perforation into the sphenoidal sinus, purulent meningitis, intradural abscess, and abscess in the frontal lobes.

[1] *Berliner Klinische Wochenschrift*, 1905.
[2] *Deutsche Medizinische Wochenschrift*, 1906.
[3] Courtaix, " Recherches Cliniques sur les Relations Patholog. entre l'Œil et les Dents," Paris, 1895.
[4] *Edinburgh Medical Journal*, 1866.
[5] *Archiv. d'Ophthalmol.*, 1885.
[6] *Archiv für Laryngologie*, 1895, Bd. III.

Westermayer[1] had a case with perforation into the pterygo-palatine fossa, caries and perforation in the large wing of the sphenoid, and an abscess in the temporal lobes.

The following conditions have been found in disease of the *frontal sinuses:* In Bousquet's[2] case perforation of the floor and posterior wall, and abscess in the frontal lobes. In Koehler's[3] case perforation of the posterior wall, intradural abscess, and basilar meningitis. In Schindler's[4] case perforation of the posterior wall, abscess in the frontal lobe, and pyæmia. In Zirm's case[5] there was thrombo-phlebitis of the orbital and cavernous veins, with orbital cellulitis and purulent emboli. In Huguénin's[6] case there was encephalitis and meningitis. In Müller's[7] case abscess over the left parietal bone, a perforation communicating with an extradural abscess, and meningitis over the right side of the convexity of the brain. In Paulsen's[8] case there was a perforation of the posterior wall of the frontal sinus, and of the dura, intradural abscess, and meningitis. In Huguénin's[9] case perforation of the posterior wall of the frontal sinus had led to purulent pachy- and leptomeningitis, intradural abscess, and fibrous adhesion between the membranes and the cerebral surface. In Knapp's[10] case there were present perforation of the posterior wall of the frontal sinus, periostitis of the orbital plate of the frontal bone, adhesion of the dura to a discoloured cerebral surface, and commencing abscess in the frontal lobes. In Bourot-Lécard's[11] case periostitis of the anterior and orbital wall of the frontal bone, thrombo-phlebitis of the orbital veins and of the cavernous sinus, and purulent meningitis were found; and in Redtenbacher's[12] case perforation of the posterior wall of the frontal sinus, with intradural and frontal abscess. In Lennox Browne's[13] case, with perforation of the posterior wall of the frontal sinus, there were combined pachy- and lepto-meningitis. In Hoppe's[14] case there was a congenital defect of the posterior wall of the frontal sinus, and meningitis of both base and convexity of the brain. In Krecke's[15] case extradural abscess and

[1] *Münchener Medizinische Wochenschrift*, 1895.
[2] *Progrès Médical*, 1877.
[3] *Charité-Annalen*, 1892.
[4] *Archives de Méd. et de Pharm.-militaires*, 1892.
[5] *Wiener Medizinische Wochenschrift*, 1892.
[6] *Korrespondenzblatt der Schweizer Arzte*, 1882.
[7] *Wiener Klinische Wochenschrift*, 1895.
[8] *Hospitals Tidende*, 1861.
[9] *Loc. cit.*
[10] *Archiv für Augenheilkunde*, 1880.
[11] *Bordeaux Méd.*, 1875.
[12] *Wiener Medizinische Blätter*, 1892.
[13] *Journal of Laryngology*, vol. vii.
[14] *Klinische Monatsblätter für Augenheilkunde*, 1893.
[15] *Münchener Medizinische Wochenschrift*, 1894.

abscess in the frontal lobe, with rupture into a lateral ventricle were found. In the case of Wallenberg[1] there was perforation of posterior and inferior walls and intradural abscess. In Silex's[2] case there was perforation of the posterior and inferior walls of the frontal sinus, with cerebral abscess on both sides. In Milligan's[3] case septic thrombosis and meningitis. In E. Fränkel's[4] case thrombo-phlebitis of the superior longitudinal sinus, with pyæmia. In Macewen and Millar's[5] case thrombo-phlebitis of the superior longitudinal sinus had occurred in the neighbourhood of the frontal sinus, and also extra- and intradural abscess, purulent meningitis, and pyæmia. In Roth's[6] case abscess of the tissues over the frontal bone, purulent meningitis, encephalitis on the right side, and abscess in the frontal lobe on the left side. Carver[7] reports thrombosis of the superior longitudinal sinus, with meningitis over the convexity of the brain, and Cholle[8] reports two cases of meningitis.

In disease of the *ethmoidal cells*, Begbie[9] reports perforation of the lamina cribrosa of the ethmoid, and abscess of the frontal lobe. Schäffer[10] reports perforation of the lamina papyracea, orbital periostitis, and perforation of the orbital roof, with an abscess in the frontal lobe. Pauncz[11] and Jacubasch[12] found cerebral abscess associated with ethmoidal disease, and Ogston,[13] Warner,[14] and Ewald,[15] found meningitis. Trousseau[16] had a case with necrosis of the ethmoid and meningitis, and Hajek[17] had one with meningitis.

In suppuration of the *sphenoidal sinuses*, Horner[18] had a case, with blindness and exophthalmos on the left side, which on post-mortem examination showed caries of the basi-sphenoid and neighbouring parts. Panas[19] reports a case which, as a result of osteitis of the sphenoid, had orbital phlegmon, exophthalmos, and blindness. In Rouge's[20] case there were present divergent strabismus, and loss

[1] *Neurologisches Zentralblatt*, 1895.
[2] *Berliner Klinische Wochenschrift*, 1896.
[3] *The Medical Chronicle*, 1899.
[4] *Virchow's Archiv*, Bd. CXLIII.
[5] Killian, "Verein Süddeutscher Laryngologen," 1900.
[6] *Wiener Klinische Wochenschrift*, 1899.
[7] *British Medical Journal*, 1883.
[8] *Archiv. Internat. de Laryng.*, 1906.
[9] *Medical Times and Gazette*, 1852.
[10] *Prager Medizinische Wochenschrift*, 1883.
[11] *Archiv für Laryngologie*, Bd. XIII.
[12] *Berliner Klinische Wochenschrift*, 1875.
[13] *British Medical Journal*, 1885.
[14] *Ibid.*
[15] *Deutsche Medizinische Wochenschrift*, 1890.
[16] *Cliniq. Médic. Deutsche*, 2 Aufl., 1866.
[17] *Archiv für Laryngologie*, Bd. XVIII.
[18] *Klinische Monatsblätter für Augenheilkunde*, 1863.
[19] Soc. de Chir. de Paris, 1873.
[20] *L'Union Médic.*, 1872.

of vision on the left side, and on post-mortem examination a purulent periostitis associated with suppuration in the left sphenoidal sinus was found. In Raymond's[1] case blindness and exophthalmos were present on both sides, and on post-mortem examination it was found that there was a purulent basal meningitis which involved the optic chiasma, along with thrombosis of the cavernous sinus and of the ophthalmic veins, due to caries and perforation in the roof of the sphenoid. In Ortmann's[2] case there was an extradural abscess, situated in the sella turcica, also thrombosis of both cavernous sinuses, and periostitis and suppuration in both sphenoidal sinuses. In Grünwald's[3] case there was basilar meningitis, and pus in the sphenoidal sinuses and maxillary antra. Vince[4] reports perforation in the basilar apophysis with meningitis ; Thiroloix,[5] extradural abscess in the sella turcica, and meningitis ; Flatau,[6] perforation of the posterior wall of the sphenoidal sinus, and meningitis ; Zöckendörfer,[7] meningitis ; Pekastovsky,[8] necrosis in the sella turcica, and meningitis, along with thrombosis of the longitudinal sinus. In Scholz's[9] case were found purulent periostitis and perforation over the upper lateral wall of the sphenoidal sinus, with meningitis, erosion of the cavernous sinus, thrombosis of the carotic sinuses and right petrosal sinus, and intradural abscess in the middle cranial fossa. In Schlagenhaufer's[10] case there was softening of the sphenoidal body, meningitis, and thrombo-phlebitis of the longitudinal sinus. In Pauncz's[11] case purulent thrombo-phlebitis of the circular sinus of Ridlei and of the inferior and superior petrosal sinuses was found. In Halász's[12] case, suppuration in the sphenoidal sinus and encephalitis. In Schroeder's[13] case, empyema of the sphenoidal sinuses, perforation, meningitis, and thrombo-phlebitis of the cavernous and petrosal sinuses.

In *multiple accessory* sinus suppurations the following conditions have been reported : Weichselbaum[14] had a case of suppuration of the right maxillary antrum and frontal sinus, with abscess over the upper lid, and extradural and frontal lobe abscesses. Grünwald[15] reported a

[1] *Bull. Soc. Anatom.*, Paris, 1895.
[2] *Loc. cit.*
[3] "Die Lehre von den Naseneiterungen," 1893.
[4] Tod, *La Clinique*, 1900.
[5] *Bull. Soc. Anat.*, 1892.
[6] *Nasenkrankheiten*, etc., 1895.
[7] *Prager Mediz. Wochenschrift*, 1893.
[8] *Internat. Zentralblatt für Laryngologie*, Bd. X.
[9] *Berliner Klinische Wochenschrift*, 1892.
[10] *Wiener Klinische Wochenschrift*, 1899.
[11] *Zeitschrift für Augenheilkunde*, 1906.
[12] *Orvosi Hetilap.*, 1904.
[13] *Zeitschrift für Ohrenheilkunde*, 1906.
[14] *Wiener Medizinische Wochenschrift*, 1890.
[15] *Loc. cit.*

case of maxillary and sphenoidal sinus suppuration, with purulent periostitis and softening in the posterior walls of both cavities. Pauncz[1] reported a case with suppuration in the maxillary antrum and ethmoidal cells on the left side, and perforation from the maxillary antrum into the orbital cavity, resulting in basilar meningitis and brain abscess. In Hansberg's[2] case, suppuration of the right frontal sinus and of all the ethmoidal cells led to caries of the ethmoid, meningitis, and abscess in the left frontal lobe of the cerebrum. Duplay[3] had a case with exophthalmos on the right side, total blindness, and basal meningitis, which was chiefly present in the sella turcica. A purulent phlebitis was found in most of the venous sinuses, and in the orbital cellular tissue there were a number of purulent areas. Pus lay in the sphenoidal sinuses, and in the right ethmoidal cells. In Russel's case[4] vision gradually diminished, and ptosis of the upper lid and immobility of the eye on the left side occurred. Post-mortem it was found that an intradural abscess lay in the middle cranial fossa, and that there was an empyema both in the sphenoidal sinus and in the right ethmoidal cells; the cavernous and petrosal sinuses and the left ophthalmic veins were thrombosed. In Vossius'[5] case, in which suppuration of the sphenoidal sinuses and the left ethmoidal cells, as well as caries of the sphenoid, had occurred, a thrombo-phlebitis of the superior ophthalmic, angular, and left anterior facial veins set in, which led to purulent pachy- and leptomeningitis. In Holmes's[6] case fronto-ethmoidal, along with sphenoidal sinus suppuration, led to diplopia, double optic neuritis, and abscess in the right frontal lobe. In a case of Demarquay's[7] the eye was immobile and proptosed, and insensitive to light on the right side. On post-mortem examination the sphenoidal, ethmoidal, and maxillary sinuses were found filled with pus, and the cavernous venous sinus contained a purulent thrombus. Panse[8] reports a case of frontal, ethmoidal, maxillary, and sphenoidal sinus suppuration, in which the cavities contained tuberculous granulations. A neuro-retinitis was present on both sides. In the ethmoid and anterior part of the sphenoid tubercular caries was present, and both orbital roofs were carious and were covered with cheesy material. Localized meningitis, internal

[1] *Orvosi Hetilap.*
[2] *Zeitschrift für Ohrenheilkunde*, Bd. XLIV.
[3] *Arch. Gén. de Méd.*, 1874.
[4] *Medical Times and Gazette*, 1878.
[5] *Zeitschrift für Augenheilkunde*, 1900.
[6] *Archives of Ophthalmology*, 1886.
[7] M. Mackenzie, "Diseases of the Nose," etc., 1884.
[8] *Archiv für Laryngologie, loc. cit.*

hydrocephalus, and caseous pneumonia, resulted. Leber[1] reports a case with tumour in the nasal cavities, empyema of the sphenoidal sinus, and blindness due to atrophy of the optic nerves. Purulent meningitis and thrombo-phlebitis of the cerebral sinuses were found on post-mortem examination. Finlag[2] reports a case with suppuration of the sphenoidal sinuses and ethmoidal cells, in which thrombo-phlebitis of the cavernous and circular sinuses occurred.

The path of infection in these cases may be by direct continuity, by the lymphatic system, or by the *venous circulation*. That the complications were due to the accessory sinus suppuration the post-mortem examinations demonstrated. It is possible that intracranial complications may arise by infection of the lymphatic channels, and especially by those that pass along with the olfactory nerves through the cribriform plate. This has not yet been proved bacteriologically or microscopically. That such disease processes as occur in the accessory sinuses, and their spread from there to the orbital contents, and to the optic nerve within its canal, may be intimately associated with the system of veins is certain. Also the intracranial and cerebral complications stand closely related to the venous system. We may include under the term "circulatory interference" such symptoms as hyperæmia, œdema of the tissues and of the optic nerve and its sheath, hæmorrhages, emboli, thrombo-phlebitis, and thrombosis. Zuckerkandl[3] has shown by his investigations that the veins of the nasal mucous membrane anastomose with those of the face.

The anterior ethmoidal veins communicate with the veins of the dura and pia mater, and the venous trunks that arise from the nasal mucous membrane anastomose with the veins of the palate and pharynx and with the pterygo-palatine plexus. The venous plexus around the lachrymal duct and the lachrymal sac opens into the anterior facial vein, and is in communication with the ophthalmic and infra-orbital veins. Zuckerkandl has described a venous twig, the lachrymo-facialis, which takes origin in the anterior ethmoidal cells and passes through the lachrymal bone. A twig also brings the plexus in the maxillary antrum into communication with the vena ophthalmica facialis. Kuhnt[4] describes anastomoses between the veins of the dura mater and those of the frontal sinus by means of perforating veins.

[1] *Archiv für Laryngologie*, Bd. XVII.
[2] *Monatsschrift für Ohrenheilkunde*, 1905.
[3] *Loc. cit.*
[4] "Entzündliche Erkrankungen der Stirnhöhle," 1895.

Kuhnt and Gurwitsch[1] also found communicating twigs between the frontal sinus veins and the superior ophthalmic and supra-orbital veins. Into the superior ophthalmic veins, too, enter the anterior and posterior ethmoidal veins, while in addition the anterior ethmoidal vein may communicate with the supra-orbital and with the angular vein. According to Ónodi,[2] the vena ethmoidalis anterior may take its course in a canal, the semicanalis ethmoidalis, through the frontal sinus and first and second orbital cells, in which it lies freely exposed for a considerable part of its course. The superior ophthalmic vein is not only in communication with the ethmoidal veins, but also with the vena centralis of the retina and with the inferior ophthalmic vein.

The orbital veins are thus in communication with the veins of the face and of the nasal cavity, and with the pterygoid plexus. They empty themselves posteriorly into the cavernous sinus. The veins of the sphenoidal sinus may also anastomose with the orbital veins and with the cavernous sinus. Sometimes the central vein of the retina opens directly into the cavernous sinus, but it may also pass through the spheno-maxillary fissure into the pterygo-palatine plexus. The inferior ophthalmic vein may also communicate with the plexus in the pterygo-palatine fossa. With so complex a system of anastomoses the disease of nasal accessory sinuses, when affecting a vein, may readily spread to neighbouring parts.

Orbital cellulitis or disease of the mucous membrane covering the veins may be the cause of a thrombo-phlebitis. Besides spread of the infection by direct extension, emboli may be set free through softening of the thrombi. Pressure from orbital cellulitis and from disease of the sheath of the optic nerve may produce congestion and œdema. In three cases of maxillary antrum suppuration, Kuhnt saw embolus of the central vein of the retina occurring. Operation was refused in these cases. In maxillary antrum suppuration, too, Fouchet observed a thrombo-phlebitis of the pterygoid and ophthalmic plexuses. In suppurations of the frontal sinuses the following observations have been made. Zirn found a thrombo-phlebitis of the ophthalmic veins and of the cavernous sinus. Bourot-Lécard observed a similar sequel. E. Fränkel, Macewen-Millar, and Carver, record thrombo-phlebitis in the superior longitudinal sinus. In sphenoidal sinus suppuration, Raymond records a case in which thrombosis of the ophthalmic veins and of the cavernous sinus occurred. Ortmann had a case with

[1] *Graefe's Archiv*, Bd. XXIX. [2] *Archiv für Laryngologie, loc. cit.*

thrombosis of both cavernous sinuses; Pekastovsky, a case with thrombosis of the longitudinal sinus; Scholz, a case with thrombosis of the carotic and right petrosal sinuses; Schlagenhaufer, thrombo-phlebitis of the longitudinal sinus; Pauncz, purulent thrombo-phlebitis of the sinus circularis Ridlei and the inferior and superior petrosal sinuses; Schröder, thrombo-phlebitis of the cavernous and petrosal sinuses.

In cases with multiple accessory sinus suppuration the following observations have been made: In Duplay's case ethmoidal and sphenoidal suppurations led to purulent thrombo-phlebitis of the majority of the venous sinuses within the cranium. In Russel's case, also with ethmoidal and sphenoidal disease, the cavernous and petrosal sinuses were thrombosed, as well as the ophthalmic veins on the left side. In Vossius' case, with disease of the ethmoidal and sphenoidal sinuses, thrombo-phlebitis of the superior ophthalmic, angular, and left facial veins occurred. Finlag, in a case of ethmoidal and sphenoidal suppuration, reports thrombo-phlebitis of the cavernous and circular sinuses.

Dehiscences in the bone walls of the accessory sinuses have an important bearing on the spread of the disease from any such sinus. Such defects in the bone may be congenital or due to senile atrophy. By the presence of such defects the mucous membrane of the frontal and maxillary sinuses may touch the periosteum of the external surface of the frontal and maxillary bones, and the periosteum of the orbital cavity. The mucous membrane of the frontal and sphenoidal sinuses may in this manner, too, be in touch with the dura mater. The membrane lining the semicanalis ethmoidalis may bring the mucous membrane of the frontal sinus and orbital cells into communication with the orbital periosteum externally and with the dura mater internally. Dehiscences in the walls of the optic canal, and of the ethmoidal and sphenoidal sinuses, may bring the mucous membrane lining the sinuses into touch with the sheath of the optic nerve. A defect in the lamina papyracea will bring the mucous membrane of the ethmoidal cells into touch with the periosteum of the orbit.

Zuckerkandl and I have observed extreme thinning of the walls of the maxillary antrum, with smaller or larger defects in parts, that have resulted from senile atrophy. In one of Zuckerkandl's cases the frontal sinus communicated with the cranial cavity, and in Merlin's case the frontal sinus communicated with the orbital cavity. Periostitis, peri-

neuritis optica, neuritis, and optic atrophy, and other sequelæ, may easily occur in such cases as have defects in the optic canal; and in defects of the cranial floor, pachymeningitis, extradural abscess, and cerebral abscess, may result. Bone defects between the accessory sinuses and the face, and pterygo-palatine fossa, may produce periostitis, cellulitis, thrombo-phlebitis, and abscesses.

Similar processes may ensue where a defect involves the walls of the orbital cavity, and in addition emphysema may result. Such unfavourable complications occur, as a rule, without the presence of bone defects. Periostitis and osteitis, leading to caries and necrosis, with perforations, occur. In such a sequence the neighbouring soft tissues may be attacked. The involvement of the veins in conjunction with or apart from such direct extension of the disease has already been considered. In this extension of disease from suppurating accessory sinuses, the optic nerve may be affected by direct involvement of the optic canal and the optic sheath, or by a periostitis which extends from the orbital cavity. The nerve may also be affected by direct pressure, or pressure on its nutrient arteries caused by orbital cellulitis, or it may be diseased secondarily to a thrombo-phlebitis of the ophthalmic veins.

Further, it must be remembered that one suppurating accessory sinus often directly affects another. Thus frontal sinus disease may extend to the orbital cells of the ethmoid, and so to the other ethmoidal cells. A communication may exist congenitally in the septum separating the two frontal sinuses, or such a communication may be due to perforation caused by inflammation. Such an intercommunication may also occur between the sphenoidal sinuses as a result of disease. Similarly, any of the thin septa that may lie between different sinuses are liable to perforation as a result of suppurative osteitis, caries, and necrosis. In this manner neighbouring sinuses are involved. Thus the maxillary antrum may infect the ethmoidal cells, and these the sphenoidal sinus, or else from the frontal sinus the advance may be in the direction of the ethmoidal cells and sphenoidal sinuses, or it may be in the reverse direction.

Besides the complications and symptoms already mentioned, it will be well to summarize such symptoms as have been recorded in accessory sinus disease. Taking the maxillary antrum first, we must mention as symptoms lachrymation, blepharospasm, and such reflex neuroses as asthma and angina pectoris, purulent dacryocystitis, and lachrymal fistula.

Nolthenius[1] records a case of rupture into the nasal tear duct of a suppurating maxillary antrum, and Treitel[2] mentions in disease of the same sinus the formation of granulation tissue and a sequestrum which led to dacryocystitis and the formation of a lachrymal fistula. Asthenopia has been recorded by Grünwald[3] and Caldwell,[4] and others.

Ziem and Fromaget state that after operating on a diseased maxillary antrum an iritis healed, and Kuhnt describes improvement of an iritis under similar conditions. Ziem and Kuhnt also saw improvement in visual power in cases of cataract in which the maxillary antrum disease had been treated. Kuhnt also observed a neuro-retinitis resolve under such treatment.

Grossmann[5] records hyperæmia of the optic disc, and Depaques[6] observed blurring of the edges of the optic disc, with diminution of visual power, due to maxillary antrum suppuration. Courtaix and Kolarowitsch[7] report amblyopia and amaurosis of dental origin.

As a result of œdema of the retrobulbar cellular tissue, exophthalmos, visual disturbance, and even blindness, due to direct pressure and to pressure on the bloodvessels of the optic nerve, may occur. Körner,[8] Le Fort,[9] and Pagenstecher,[10] record exophthalmos. Passing amblyopia or amaurosis has been observed by Brück,[11] Pasquier,[12] Buzer,[13] Galezowski,[14] and Ónodi.[15] Grünwald[16] and Panzer[17] have, as a result of perforation of the wall of the maxillary antrum, seen an abscess and fistula in the hard palate. Tedenat[18] and Westermayer[19] have seen cases with phlegmon of the pterygo-palatine fossa. Bournonville,[20] Jansen,[21] Flatau,[22] and Lichtwitz,[23] have described cases in which rupture occurred through the wall of the inferior meatus, and Killian[24]

[1] *Monatsschrift für Ohrenheilkunde*, 1895.
[2] *Verhandlungen der Laryngologischen Gesellschaft*, Berlin, 1900.
[3] *Loc. cit.* [4] *Medical Journal of New York*, 1898.
[5] Weinlechner, *Bericht der k. k. Krankenanstalt Rudolfstift.*, 1875.
[6] *Bull. de la Soc. d'Ophth. de Paris*, 1893. [7] *Loc. cit.*
[8] *Verhandlungen der Deutschen Odontologischen Gesellschaft*, Bd. VII.
[9] *France Méd.*, 1876. [10] *Archiv für Augenheilkunde*, 1884.
[11] *Wochenschrift für die Gesamte Heilkunde*, Berlin, 1851.
[12] *Lanc. Franç.*, 1839. [13] *Berliner Klinische Wochenschrift*, 1868.
[14] *Archives Intern. de Larnyg.*, 1905.
[15] *Revue Hebdomadaire de Laryng.*, etc., 1905.
[16] "Lehre von der Naseneiterung," 2 Aufl.
[17] *Wiener Klinische Wochenschrift*, 1896.
[18] Soleville, *Thèse de Montpelier*, 1890.
[19] *Münchener Medizinische Wochenschrift*, 1895.
[20] *Zentralblatt für Chirurgie*, 1885. [21] *Archiv für Laryngologie*, Bd. I.
[22] *Rev. Int. de Rhin.*, 1893. [23] *Annal. de Malad. d'Or.*, 1896.
[24] *Heymann's Handbuch*, Bd. III.

records a case of perforation through the middle meatus. Dmochowski[1] saw a case in which a suppurating maxillary antrum ruptured into the sphenoidal sinus. Bauby,[2] Dmochowski,[3] Kuhnt,[4] Salva,[5] and Fischer,[6] report cases in which rupture occurred into the orbital cavity. Orbital cellulitis, orbital abscesses, and thrombo-phlebitis with intracranial complications may, as already mentioned, also occur in similar cases.

In frontal sinus suppuration, reflex neuroses, such as bulbar and periorbital neuralgias, have been observed, and but rarely dacryocystitis. Kuhnt and Lichtwitz have described hyperæmia of the optic disc, with fulness of the retinal veins, and a retinal infiltration around the disc ; and Kuhnt, Ónodi, and others, have seen cases with exophthalmos and diplopia. Schmiegelow[7] and Jacqueau[8] have observed exophthalmos of rapid onset. Kuhnt has also noticed a haziness in the lens. Lapersonne[9] has observed transitory or permanent pareses of the nerves of the ocular muscles.

Periostitis, perforation of the walls of the frontal sinus, œdema of the retrobulbar tissue, orbital cellulitis, and abscess—these have been frequently recorded. Bourot, Lécard, Carver, and Schröder, have seen subperiosteal abscess on the orbital roof, and also caries and necrosis. Spencer Watson[10] removed two sequestra, and Panas[11] one, from the orbital plate of the frontal bone. Bäumler,[12] Steinthal,[13] Köhler,[14] Roth, and Macewen-Millar, have observed periostitis of the anterior wall of the frontal sinus, cellulitis of the forehead and upper lid, and abscess formation in these parts ; and Botey[15] had a case in which a perforation of the anterior wall of the sinus occurred. The anterior wall is less liable to disease than the inferior wall. Cases have already been referred to in which the posterior wall of the frontal sinus was perforated, and others in which orbital abscess, thrombophlebitis, intracranial and cerebral complications, occurred.

Burger,[16] Caldwell,[17] Hajek,[18] and Grünwald,[19] have seen cases with

[1] *Archiv für Laryngologie*, Bd. III.
[2] *Arch. d'Ophthalm.*, 1897.
[3] *Loc. cit.*
[4] *Loc. cit.*
[5] *Thèse de Paris*, 1895.
[6] Salva, *loc. cit.*
[7] *Archiv für Laryngologie*, Bd. XV.
[8] *Internationales Zentralblatt für Laryngologie*, Bd. XVI.
[9] *Bull. de la Société Franç. d'Ophthal.*, 1902.
[10] "Diseases of the Nose and its Accessory Cavities."
[11] *Progrès Méd.*, 1887.
[12] Kongress für Innere Medizin, 1890
[13] *Medizin. Korrespondenzblatt der Württembergischen Ärztlichen Landesvereine*, 1891.
[14] *Charité-Annal.*, 1891.
[15] *Revue de Laryng.*, 1897.
[16] *Zentralblatt für Laryngologie*, Bd. XI.
[17] *Ibid.*, Bd. X.
[18] "Erkrankungen der Nasenhöhlen der Nase," 1903.
[19] *Loc. cit.*

asthenopia, due to ethmoidal suppuration. Such conditions as retrobulbar neuritis and choked disc will be referred to later. Purulent dacryocystitis is a recognized complication of ethmoidal suppuration. The eye may be pushed aside by a mucocele of the ethmoidal cells or by an empyema.

Hjorth[1] and Ónodi[2] observed exophthalmos in cases of ethmoidal mucocele without disturbance of vision. Baurowitz[3] has seen diplopia with exophthalmos in such cases, and Mann[4] has observed optic neuritis and exophthalmos. Richet[5] has seen a similar case leading to blindness. In a patient of Schmiegelow[6] the mucocele pushing the eye aside contained sero-purulent fluid. Eversbusch[7] has observed a case of ethmoidal mucocele in which the exophthalmos was intermittent. Those complications that lead to perforation of the orbital plate of the ethmoid and produce orbital cellulitis have already been referred to, and also such as lead to intracranial complications.

Baumgarten[8] observed the presence of oculo-motor paresis in a case of sphenoidal suppuration, and Post[9] has seen incurable blindness and exophthalmos as a result of necrosis of the small wing of the sphenoid. In other respects the patient had been cured. Orbital cellulitis may also occur in cases of sphenoidal suppuration. The cases that pass on to intracranial infection have already been referred to. Choked disc, retrobulbar neuritis, and optic atrophy, will be discussed later in detail as complications of sphenoidal suppuration.

In a case recorded by Sokos and Luc,[10] maxillary and frontal sinus suppuration was associated with diplopia, and in a case of maxillary and sphenoidal sinus suppuration Lapersonne[11] observed total paralysis of the oculo-motor nerve. Ingersoll described a case of frontal sinus and ethmoidal cell suppuration, in which exophthalmos was present; and in a similar case Broeckart[12] observed vitreous haziness, with detachment of the retina. Hoffmann[13] in one case found rupture into the orbital cavity, and an orbital abscess, associated with frontal, ethmoidal, and sphenoidal sinus suppuration; and in another case of ethmoidal and sphenoidal suppuration he observed exophthalmos and diplopia.

[1] Schmiegelow, *Archiv für Laryngologie*, Bd. XV.
[2] *Archiv für Laryngologie*, Bd. XVII. [3] *Ibid.*, Bd. XII.
[4] *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1901.
[5] Kuhnt, *loc. cit.* [6] *Archiv für Laryngologie*, Bd. XV.
[7] "Graefe-Sämisch' Handbuch," 2 Aufl. [8] *Orvosi Hetilap*, 1903.
[9] *Lancet*, 1882. [10] *Revue de Laryngol.*, 1899. [11] *Loc. cit.*
[12] *Revue de Laryngol.*, 1901.
[13] *Zeitschrift für Augenheilkunde*, 1906.

F. R. Alexander[1] has seen a case in which a metastatic abscess in the vitreous of one eye was associated with suppuration in the maxillary antrum and ethmoidal cells.

I proceed next to the subject of neuritis of the optic nerve trunk within its canal, and of optic atrophy. As has already been pointed out, inflammatory disease of the orbital tissues may spread directly to the optic nerve. This, and also pressure on that portion of the optic nerve that is free from bloodvessels, as well as pressure on its nutrient bloodvessels, may lead to visual disturbances and to blindness. In a similar way disease of those nasal accessory sinuses which are in close proximity to the nerve may extend through the optic canal to the sheath of the optic nerve. Any bone defect in intervening septa between such sinuses and the optic nerve will favour such a direct extension of the inflammation.

Direct infection through intervening bone is by osteitis and periostitis, though it may also be by venous thrombosis. Duplay, Horner, Panas, Rouge, Russel, Raymond, Demarquay, Vossius, Holmes, and others, have observed cases in which visual disturbance or blindness was associated with disease of nasal accessory sinuses, and in which the diagnosis was confirmed by post-mortem examination. Although in some cases optic atrophy had already occurred, yet in others treatment of the diseased nasal sinuses led to improvement, and even cure, of the optic condition. On the other hand, in similar cases of associated visual disturbance and sinus disease, treatment of the sinuses at fault led to no improvement of the optic disease.

Our knowledge of the path of infection, and of the optic nerve, its sheath and bloodvessels, are as yet insufficient. We are also still in want of detailed records of the anatomical relations of the accessory sinuses to the optic nerve in these cases.

Even in cases of caries and necrosis of the sphenoidal sinus walls there may be no optic neuritis. Berger and Tyrmann[2] have recorded a case of gradual separation of a sequestrum from the body of the sphenoid which finally led to meningitis without producing at any time signs of visual disturbance. Baratoux[3] describes a case in which a sequestrum separated from the sphenoid, and was removed through the nose without causing any complications.

Hajek[4] has observed extensive destruction of the anterior wall of the sphenoid produced by syphilis, which caused no exceptional

[1] *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1905.
[2] *Loc. cit.*
[3] *Arch. Ital. di Laryng.*, 1883.
[4] *Loc. cit.*

symptoms. Flatau[1] records twenty-six cases of sphenoidal sinus suppuration with caries, yet showing no symptoms of ocular disturbance. Foucher[2] describes necrosis of turbinated bones and of the wall of the sphenoidal cavity in the case of a girl fifteen years of age, in whom mercury as an antisyphilitic remedy was given. Sequestra finally separated, and the child died. At no time were visual disturbances detected.

Schäffer,[3] Ónodi,[4] Schmiegelow,[5] and Hoffmann,[6] also state that in numerous cases of sphenoidal suppuration no changes in the visual power can be detected. Hinkel[7] treated twenty cases of sphenoidal suppuration without finding anything abnormal in the visual fields or in the ocular fundi. Contrary to the findings of Grünwald,[8] Ziem,[9] Berger,[10] Kuhnt,[11] and Bryan,[12] Henrici and Häffner,[13] in thirty-six cases of accessory sinus disease, found a normal visual field. These facts are to be explained, and in this Hoffmann[14] agrees, by the varying relation of accessory sinuses and optic nerves, as already described. There are two factors that help in the limitation of accessory sinus disease, and prevent it from spreading to the optic nerves: the nerves may come into no relation with the sphenoidal sinuses or ethmoidal cells, and the wall of the sphenoid may be very thick. The variation in thickness ranges between 1 millimetre and 12 millimetres.

It is evident that even destruction of bone may occur in accessory sinuses, and yet no visual disturbance result.

In considering optic neuritis, we must limit ourselves to its association with accessory sinus disease. It is necessary to point out that the stereotyped explanation that such an optic neuritis is due only to sphenoidal sinus suppuration cannot be maintained. As I have already pointed out[15], the optic nerves may be in close proximity to the ethmoidal cells, and yet be in no relation to the sphenoidal sinuses. Schmiegelow,[16] Alexander,[17] and Hoffmann,[18] have already taken cognizance of this fact, and with regard to that part of the nerve which lies in the optic canal Pauncz[19] accepts the same view; but he

[1] *Zentralblatt für Laryngologie*, 1894-1895. [2] *Ibid.*
[3] *Deutsche Medizinische Wochenschrift*, 1892.
[4] *Archiv für Laryngologie, loc. cit.* [5] *Loc. cit.* [6] *Loc. cit.*
[7] American Laryng. Assoc., 1902. [8] *Loc. cit.*
[9] *Berliner Klinische Wochenschrift*, 1888.
[10] "Rapports entre les Malad. des Yeux," etc., 1892. [11] *Loc. cit.*
[12] American Laryng. Assoc., 1895.
[13] *Münchener Medizinische Wochenschrift*, 1904. [14] *Loc. cit.*
[15] *Zeitschrift für Augenheilkunde, loc. cit.; Archiv für Laryngologie, loc. cit.*
[16] *Loc. cit.* [17] *Loc. cit.* [18] *Loc. cit.*
[19] *Archiv für Augenheilkunde*, Bd. LII.

holds that the intracranial portion of the nerve which lies between chiasma and optic canal lies in close relation solely with the sphenoidal cavity, and from that it is separated by thick bone. These two statements can be controverted by the anatomical relations already detailed. Where visual disturbance is associated with suppuration both in sphenoidal sinuses and ethmoidal cells, we are not correct in taking into consideration only the sphenoidal sinus suppuration. Schmiegelow[1] takes up the right attitude in considering both the ethmoidal and sphenoidal suppuration as factors, without discriminating between them. Halstead[2] and Pollatschek[3] have in cases of sphenoidal and ethmoidal suppuration, associated with visual disturbance, considered only the sphenoidal suppuration as a causal factor, without giving any reason for excluding from consideration the disease in the ethmoidal cells.

Spreading of the disease, whether by direct extension or through affection of bloodvessels, is just as possible from the posterior ethmoidal cells as from the sphenoidal sinuses.

Mendel[4] and Lapersonne[5] state that a one-sided optic neuritis, broadly speaking, is referable to intranasal disease. Mendel found that in about one-half of the cases of unilateral optic neuritis this relation to intranasal trouble could be made out. As double optic neuritis is characteristic of intracranial trouble, so unilateral optic neuritis, he says, is characteristic of orbital disease. Optic neuritis, he says, is rarely caused by frontal sinus disease, more often by maxillary and ethmoidal disease, but chiefly by sphenoidal disease. The main characteristic of an optic neuritis purely of accessory sinus origin is that it is unilateral. Although, strictly speaking, it is possible for both optic nerves to be affected in their canals by double sphenoidal suppuration, yet a double œdematous neuritis should specially make us suspect an intracranial process.

Professor H. Sattler,[6] on being approached by me on this subject, answered that unilateral optic neuritis, and also optic atrophy, are by no means characteristic of disease of the sphenoidal sinuses or ethmoidal cells alone. Inflammations, hæmorrhages, or tumours, at the cerebral end of the optic canal, or toxic agents, may similarly cause a unilateral neuritis. The rule laid down by Mendel and Lapersonne cannot be maintained, for against its application is every case in which

[1] *Loc. cit.* [2] American Laryng., Rhin., and Otol. Society, 1901.
[3] *Zentralblatt für Laryngologie*, Bd. XXII.
[4] *Zentralblatt für Augenheilkunde.*
[5] *Loc. cit.* [6] Ónodi, *Archiv für Laryngologie*, Bd. XVI.

double visual disturbance occurs associated with unilateral sinus suppuration. And such a double affection arising from disease of the posterior ethmoidal cell or sphenoidal sinus of one side is possible, as can be proved by the morphological observations already indicated by me. In this Mendel and Lapersonne can be supported—that the majority of visual disturbances caused by accessory sinus disease have been unilateral.

Less in number are the cases of bilateral sinus suppuration associated with double optic neuritis, and rarer still cases of double optic neuritis caused by unilateral sinus suppuration, or a contralateral optic neuritis caused by one-sided sinus suppuration. As an ætiological factor must not be forgotten those cases of visual disturbance caused by operative procedure on the accessory sinuses.

This occurred in a case observed by Hirschberg.[1] An operation was carried out through the maxillary antrum in order to open up the ethmoidal and sphenoidal sinuses on the right side, on account of suppuration in all these sinuses. After the operation the upper half of the pupil showed insufficiency of action. This must have been caused by a lesion of the optic nerve.

As has already been noted—and a glance at Figures 9, 10, and 31, will recall it—the optic nerve may for a varying distance run in the wall of the sphenoidal sinus or posterior ethmoidal cell.

Of cases of sinus suppuration on one side causing visual disturbance or blindness on that side, we have already mentioned the records of Duplay, Horner, Panas, Rouge, Russel, Raymond, Demarquay, Leber, and Holmes. Berger[2] records two cases of optic neuritis that ended in amaurosis. Grossmann[3] assumes that the association between disease of the sphenoidal sinus and visual disturbance in four of his cases was a causal and not an accidental one. Snellen,[4] in two cases of optic atrophy, ascribes the change to sphenoidal suppuration, and Knapp[5] refers optic disc changes in one case to disease of the sphenoidal sinus. Hirschmann believes the cause of a temporal hemianopsia, atrophic changes in the temporal half of the left disc, and pallor of the similar part of the right disc, in a case of his, to have been either suppuration in the sphenoidal sinuses or a tumour in the region of the sella turcica. This case was not operated upon, and did not come to post-mortem examination.

In the above cases more detailed information is lacking.

[1] *Zentralblatt für Augenheilkunde.* [2] *Loc. cit.*
[3] *Allgemeine Wiener Medizinische Zeitung*, 1893.
[4] *Ned. Tijdschr. vor Geneesk.*, 1894. [5] *Zeitschrift für Ohrenheilkunde*, 1894.

Lapersonne[1] reports three cases in which operation on diseased sinuses had no influence on the course of associated optic neuritis. Grónbäk[2] reports a case of amaurosis associated with ethmoidal suppuration, in which operation on the sinuses had no influence on the blindness. Post[3] records a case with necrosis of the small wing of the sphenoid, in which exophthalmos and amaurosis were present. On removal of the sequestrum, the exophthalmos disappeared, but the amaurosis remained. Risley[4] reports a case of ethmoidal and sphenoidal suppuration in which the eye became blind.

Cases are on record in which a one-sided disturbance of vision associated with accessory sinus disease got well, sometimes as a result of treatment, and sometimes without it.

Coppez and Lor[5] report a case of sphenoidal sinus suppuration in which an associated one-sided optic neuritis healed spontaneously, notwithstanding the refusal of all surgical interference. Mendel also remarks that retrobulbar optic neuritis may get well spontaneously, or after very simple means of treatment. Coppez's[6] case had suppuration of the sphenoidal sinus, with the presence of a sequestrum 1 centimetre long. The eyeball was turned outwards and a very little forwards ; the left optic disc was blurred, and vision was defective. The case was cured. Mendel[7] reports a case of one-sided retrobulbar optic neuritis which improved very much in vision after opening up diseased ethmoidal cells. Bergmeister and Hajek[8] report a case of cure of a one-sided choked disc after treatment of ethmoidal suppuration. Fliess[9] saw some benefit result from treatment in a case of right-sided sphenoidal sinus suppuration associated with retrobulbar neuritis. In Lor's[10] case of sphenoidal sinus suppuration associated with optic neuritis and symptoms of sudden blindness, operation resulted in a cure of the eye condition. Nuel[11] reports three cases similar to this one. Either neuritis or neuro-retinitis was present. Hoffmann[12] reports such a case, associated with sphenoidal and ethmoidal suppuration, in which treatment was followed by successful results. Holmes[13] cured a case

[1] *Loc. cit.*
[2] *Verhandlungen des Danischen Oto-Laryngologischen Vereines*, 1904.
[3] *Loc. cit.*
[4] *Internationales Zentralblatt für Laryngologie*, 1905.
[5] *La Presse Méd. Belge*, 1900.
[6] *Ophthalmolog. Klinik*, 1902.
[7] *Loc. cit.*
[8] Hajek, "Die Erkrankungen der Nebenhöhlen der Nase," 1903.
[9] *Wiener Klinische Rundschau*, 1895.
[10] Schmiegelow, *Archiv für Laryngologie*, Bd. XV.
[11] *Ibid.*
[12] *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1897.
[13] *Archives of Ophthalmology*, 1896.

of visual defect of the left eye by treating a left-sided sphenoidal suppuration by operative means. Pauncz[1] had a case in which, on opening up and cleansing suppurating ethmoidal cells, marked amelioration of an optic neuritis occurred. Schmiegelow[2] also obtained much improvement in the visual power of a patient by operating on a sphenoidal and posterior ethmoidal suppuration that was associated with it. In a case reported by Fuchs and Hajek[3] a retrobulbar neuritis with central scotoma was cured after removing polypoid degenerated mucous membrane from the left ethmoidal cells and sphenoidal sinuses.

Only a few cases are on record in which a double-sided accessory sinus suppuration was associated with double optic neuritis. Mendel[4] cites from an English source a case of double visual disturbance, the cause of which lay in suppuration of the ethmoidal cells. A case of double optic atrophy occurred in my practice associated with multiple sinus suppuration. Pollatschek[5] accounts for bilateral amaurosis in a case of his by the presence of sphenoidal sinus suppuration—a view supported by me in the discussion[6] that followed. F. R. Alexander[7] reports a case of double optic neuritis in which, after the opening up of suppurating ethmoidal cells on the right side, and a suppurating sphenoidal sinus on the left side, a cure was obtained. Schmiegelow[8] saw marked improvement in a case of double optic neuritis after operating on associated suppuration of the ethmoidal and sphenoidal sinuses of both sides. Delneuville[9] treated a case of sphenoidal suppuration, in which visual disturbance was present on both sides, by means of irrigation of the sinuses. After a month of such treatment the eye condition was cured.

These various observations can be explained if we consider the close relation of the optic chiasma and optic nerve to the sphenoidal sinus and most posterior ethmoidal cell. The course of the optic canal may for a greater or less distance lie actually within the sphenoidal sinus, or the ethmoidal cell, or it may lie in close proximity, with but a thin bone intervening between the nerve and the sinus cavity. The ethmoidal veins, too, not infrequently lie exposed in the semicanalis ethmoidalis.

[1] *Zentralblatt für Laryngologie*, Bd. XXII.
[2] *Archiv für Laryngologie*, Bd. XVIII.
[3] Fuchs, "Lehrbuch der Augenheilkunde," 1905.
[4] *Loc. cit.* [5] *Orvosi Hetilap.* [6] *Ibid.*
[7] *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1905.
[8] *Archiv für Laryngologie*, Bd. XVIII.
[9] *La Presse Oto-Laryngologique Belge*, 1906.

That contralateral and double optic disturbances may arise from a one-sided disease of the accessory sinuses is to be explained by the anatomical conditions that may exist, and that have already been described. According to my observations, the following anatomical relationships are possible :

1. The left most posterior ethmoidal cell forms the inner or medial wall of the right optic canal.

2. The right most posterior ethmoidal cell forms the floor and inner wall of the optic canal on both sides, and the wall of the whole optic sulcus.

3. The right most posterior ethmoidal cell forms the wall of the whole optic sulcus.

4. The right most posterior ethmoidal cell forms the wall of the right third and the middle third of the optic sulcus.

5. The left sphenoidal sinus forms the floor of the right optic canal.

6. The right sphenoidal sinus is only in relation with the left optic nerve on the inner or medial side.

7. The left sphenoidal sinus forms the floor of the right optic canal, and the wall of the right and middle third of the optic sulcus.

8. The left sphenoidal sinus forms the floor and inner walls of the optic canals on both sides, and the wall of the whole optic sulcus.

9. In this the relations are similar to those in No. 8, except that in addition the greater part of the floor and inner wall of the right optic canal is formed by the right most posterior ethmoidal cell.

10. The left sphenoidal sinus forms the floor and inner wall of the left optic canal, the floor of the right optic canal, and the wall of the whole optic sulcus.

11. The right sphenoidal sinus forms the wall of the middle third of the optic sulcus.

The presence of symptoms of unilateral visual disturbance on the side opposite to that of the disease or lesion has been recorded in but few cases. Wohlmuth[1] reports a case of traumatic blindness in the left eye, with signs of injury visible only on the right side of the forehead over the right orbital ridge, and also over the occiput where there was a horizontal wound. Berlin[2] assumes that a fracture in the right orbital roof crossed over into the left optic canal, or that the fracture was an indirect one due to the blow on the occiput. Freudenthal[3]

[1] Berlin, "Graefe-Samisch," 1 Aufl. [2] *Ibid.*

[3] *Archives Internat. Chauveaux et St. Hilaire*, Paris, 1905.

saw blindness on the left side, with obvious optic atrophy six weeks later, due to an operation by Killian's method on the right frontal sinus. The ophthalmic surgeons Oppenheimer and May take the view that in consequence of the operation an indirect fracture in the region of the left optic canal occurred. Freudenthal accepted this explanation, as no other seemed probable.

Laas and Lewy report a case in which a septal resection was carried out from the left nostril, and an abrupt spur 4 centimetres long and $2\frac{1}{2}$ centimetres broad, that passed backwards and upwards, was removed. A quarter of an hour later there was a flickering sensation in the right eye, with visual defect. Laas found a complete defect in the upper half of the visual field in the right eye. The fundus appeared normal, except for some pallor of the right disc. At the end of two weeks the optic disc showed complete pallor. My opinion being asked in this case, I said I thought it was probably a fracture of the right optic canal. With this view Laas[1] and Lewy agree. The suddenness of the onset is in favour of it.

It has been shown that the optic canal may run within the sphenoidal and ethmoidal sinuses; and also that the sphenoidal sinus may extend between the lamellæ of the nasal septum at its posterior attachment to the sphenoid. Further, the ethmoidal or sphenoidal sinus of one side may cross over the middle line. These facts, illustrated in Figures 14, 15, 67, 72, 73 (Atlas), will give an anatomical basis for the explanation of such a fracture.

Even though a fracture of the optic canal on the same side is more frequently observed, the cases just mentioned may quite possibly be explained by the occurrence of an indirect fracture. It may happen that an optic canal is separated by a very thin plate of bone from the sphenoidal sinus or posterior ethmoidal cell of the side opposite to that on which the sinuses are affected.

In explaining these cases by a fracture of the optic canal, it must necessarily be assumed that it is an indirect fracture. Hölder found fracture of the optic canal in fifty-three cases. Freudenthal's case, if correctly explained, is a unique one. It must have occurred on removal of the floor of the frontal sinus, as, in his patient, a Kuhnt operation had already been done on some previous occasion.

Neither a traumatic fracture on the same side, nor on the opposite side, involving the optic nerve, has yet been observed in any other of

[1] Laas reported the case during the February session of the Berliner Ophthalmologischen Gesellschaft.

the numerous cases of frontal sinus operation by Killian's method. Yet in the ordinary cases of Killian's operation the hammer and chisel are more freely used than in Freudenthal's already partially completed operation. Halstead[1] reported a case of right, maxillary, ethmoidal, and sphenoidal suppuration in which blindness of the left eye occurred. Halstead assumed that the left sphenoidal sinus was opened into by the disease, and that secondarily to this a left optic neuritis occurred. He then operated. As six months after the operation the eye was again normal, Brown assumes that an exudate had occurred into the sheath of the optic nerve. The ethmoidal cells might just as well have been responsible, and even in spreading the inflammation to the opposite side the ethmoidal cells may be just as active as the sphenoidal sinus.

Halstead's theory that it was due to a rupture into the left sphenoidal sinus is an unnecessary hypothesis. Glegg and Hay[2] reported a case of bitemporal hemianopsia and paralysis of association movements due to ethmoidal suppuration on the right side. They assume the lesion to be one of the optic chiasma, and of Gudden's commissure, thus accounting for paralysis of association movements. Cure of the eye lesion occurred after operating on the disease. In this case the ethmoidal cells may just as easily have been the cause of the chiasma lesion as disease of the sphenoidal sinus. Pollatschek[3] had a case of ethmoidal and sphenoidal suppuration on the left side in which double optic neuritis was present. After operative treatment cure of the neuritis occurred. In explanation, he assumes the existence of a dehiscence in the wall of one or both sphenoidal sinuses, and an extension by periostitis to both optic nerves. This he thinks most probable. Much stinking pus came from the posterior ethmoidal cells, and but little pus from the sphenoidal sinus. He gives no particulars as to the amount of expansion of either the posterior ethmoidal cells or of the sphenoidal sinus, nor does he assign any reason for excluding as a factor in the causation of the optic neuritis the disease in the posterior ethmoidal cells. The ethmoidal cells may be an important factor in such affections of the optic nerves. We must, however, bear in mind the possibility of an accidental association of the optic neuritis with the diseased sinuses.

Polyák[4] reports a case of exophthalmos and optic atrophy on both

[1] American Laryng., Rhin., and Otol. Society, 1901.
[2] *Archiv für Laryngologie*, Bd. XVII.
[3] *Zentralblatt für Laryngologie*, Bd. XXII.
[4] *Archiv für Laryngologie*, Bd. XV.

sides, in which the right eye became affected a year before the left. Bullous air-cells were present on the left side, with multiple latent accessory sinus empyemata. He assumes that the pus in the left bullous cells tracked to the sinuses of the left side, and then to those of the right. Finding no exit, it caused dilatation of the sinuses, and so pressure effects. Thus both the exophthalmos and the optic atrophy can be explained. Polyák's explanation I held was not tenable. Both Goldzieher and I propounded an explanation, but this it is needless to state now, as the condition will probably soon be cleared up by an autopsy. Gutmann[1] observed a case of blindness in the right eye that developed in association with suppuration of the left maxillary antrum. He believes that the disease extended to the ethmoidal and sphenoidal sinuses, and so crossed to the opposite side and involved the optic nerve, causing amaurosis. In my own case a suppuration of the posterior ethmoidal cells, and of the sphenoidal sinus, was associated with optic atrophy of long standing on the right side, and optic neuritis with temporal hemianopsia on the left side. The left eye improved slightly after operation, from $\frac{6}{15}$ to $\frac{6}{10}$. Six years previously many polypi had been removed from the left nasal cavity. This was followed by purulent discharge of offensive odour on that side. There was continual headache ; in two months' time the right eye became blind, and three months later the visual disturbance in the left eye commenced. When examined, the left eye showed temporal hemianopsia. There was no history of syphilis, and treatment by mercury and iodides had no effect.

In Figure 50 a diagram of the defect in the visual field is shown. The patient, recommended to me by Professor Szily, passed out of further observation, and her subsequent history is unknown to me. We agreed that there was a true ætiological connection between the accessory sinus disease and the visual disturbance. I have a specimen in which the left sphenoidal sinus bounds only the right optic canal and the middle third of the optic sulcus. In such a relation, a lesion of the right optic nerve and chiasma, leading to atrophy of the right nerve, and producing hemianopsia on the left side, might possibly be induced by sphenoidal sinus disease. An accidental association of the optic trouble with the sinus disease is also possible, but this could only be decided by post-mortem examination.

As regards the deductions from anatomical data which indicate the possibility of visual disturbance on the opposite side from sinus

[1] *Zeitschrift für Augenheilkunde*, 1906.

disease, or the occurrence of double optic neuritis from unilateral nasal disease, it must be acknowledged that they are as yet unconfirmed by post-mortem evidence.

The treatment of the individual sinuses is dealt with in detail in my book on that subject ;[1] I will only now emphasize this one point. In cases of visual disturbance in which the ophthalmic surgeon can come to no conclusion, and in which accessory sinus disease is a possible factor, the rhinologist should not rest content until he has proved that there is no latent suppuration. Even should it appear, on exploration,

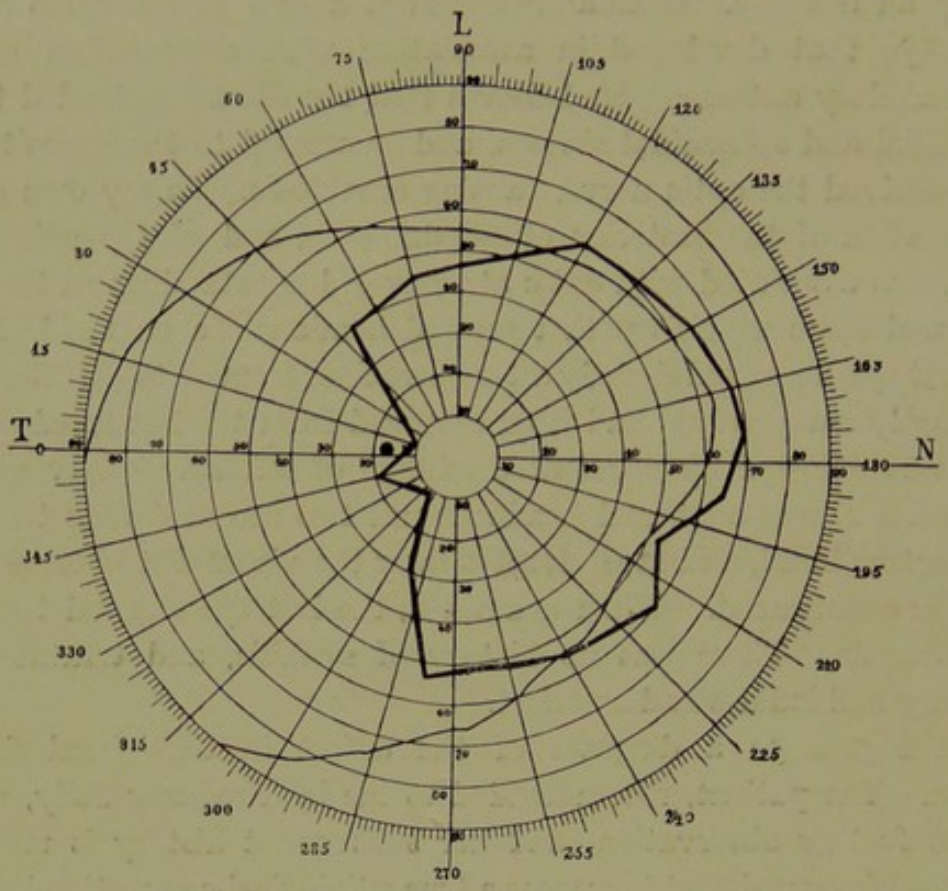


FIG. 50.

that there is no latent suppuration, this knowledge is yet of service in excluding accessory sinus disease. Schmiegelow's[2] interesting case, and the favourable results that followed, confirm this view.

In closing these observations on an interesting subject, we may take it as firmly established that accessory sinus disease, both ethmoidal and sphenoidal, may cause visual disturbance or blindness on one or both sides, or disease of the optic nerve of the opposite side. And

[1] Ónodi and Rosenberg, "Die Behandlung der Krankheiten der Nase und des Nasen-Rachenraumes," 1906.

[2] *Loc. cit.*

though our pathological knowledge is not complete, the anatomical relations that may be present suffice to explain every clinical finding. Improbable hypotheses need thus no longer be put forward to explain all the associated symptoms and complications of accessory sinus suppuration. Careful clinical observations, both by ophthalmologists and rhinologists, and, where possible, post-mortem examinations, will in time fully develop our knowledge of this important subject.

In addition to the visual disturbances already dwelt on, we have to consider another defect that has recently been receiving attention. Fuchs's observations lead him to think that acute retrobulbar neuritis with central scotoma frequently occurs after nasal catarrh and influenza. Rhinological examination may be negative. The central scotoma is due to affection of the papillo-macular bundle of the optic nerve, which runs axially. Hyperæmia and swelling of the nerve sheath induces this change in the axial nerve bundle, which seems peculiarly vulnerable. That it is not destroyed in structure is certain, because of the frequent restoration to normal function that occurs.

Accessory sinus suppuration and pressure on the nerve, or on the bloodvessels of that part which receives recurrent twigs from the central artery of the retina, and which the central vein of the retina joins, may cause a similar condition of central scotoma. Swelling within that part of the optic canal may cause venous congestion, and so injure the papillo-macular bundle. A venous congestion within the accessory sinuses and nasal cavities may likewise indirectly affect the optic nerve. The veins in the semicanalis ethmoidalis, as has already been described, may run exposed in some of the ethmoidal cells, and so be open to pressure and infection. Zuckerkandl has described a venous twig that may run exposed in some ethmoidal cells. And, as has been described, the optic nerve may be in close relation to the sphenoidal sinus and posterior ethmoidal cell; the optic canal may even lie within the cavities.

That inflammations in the sinuses, by direct extension to the nerve sheath or through veins, may cause a disturbance of the axial bundle of the optic nerve is easily comprehensible. Toxic effects, too, may produce the central scotoma. It is through its vulnerability to toxins that the lesions of the papillo-macular bundle are best known. This explains tobacco amblyopia. Birsch-Hirschfeld lays emphasis on this vulnerability. Experimentally and clinically this axial degeneration

has been proved. He believes that the presence of a central scotoma is an early and important symptom of suppuration in the posterior accessory sinuses, or of the presence of a tumour there. In a case of carcinoma with this symptom, he was able to point out an isolated axial degeneration of the optic nerve posterior to the point of entry into the nerve of the vena centralis retinæ. The new growth pressed on the central vein and caused axial degeneration, as well as separation through œdema of the neuroglia fibres and septal sheaths, and increase as well as turbidity of the neuroglia cells. This he ascribes to venous congestion and toxic œdema caused by compression of the central retinal vein by the tumour. As the periphery of the visual field was normal, the phenomena cannot be ascribed to direct pressure. We must assume that the axial degeneration was due to a toxin.

In a case of mine in which I did not test for scotoma, the optic nerve was histologically normal. Yet the nerve in its optic canal was surrounded by sarcoma. In this case the microscopic examination showed distinct pressure on, and congestion of, the central vein of the retina. The fundus of the eye was normal in appearance as seen by the ophthalmoscope. It is legitimate therefore to assume that accessory sinus suppuration may cause central scotoma in the visual field, in the same way as it occurred in Birsch-Hirschfeld's case. Birsch-Hirschfeld thinks that a one-sided visual disturbance is characteristic of accessory sinus suppuration, as contrasted with double optic neuritis of toxic origin, or such as are caused by general infections.

Complications not sufficiently referred to in describing ocular disturbances in accessory sinus suppuration are neuritis of the fifth nerve and paresis of the motor nerves of the eye muscles. Thomson observed complete oculo-motor paralysis in a case of suppuration of the ethmoidal and sphenoidal sinuses.

Hoffmann, Lapersonne, and Stanculeanu, observed oculo-motor paralysis in cases of sphenoidal sinus suppuration. Baumgarten has seen oculo-motor paralysis in accessory sinus suppuration, and Panas paralysis of the abducens and also of the fifth nerve. Mahu describes abducens paralysis, and Fisch paralysis of the adducens and abducens. Panas has seen anæsthesia of the second division of the fifth nerve; Rouge, infra-orbital neuralgia; and Schäfer, Moreau, Hajek, and Schröder, supra-orbital neuralgia. In some cases the anatomical relations of the accessory sinuses render such complications possible.

The sphenoidal sinuses and the posterior ethmoidal cells may extend into the large and small wings of the sphenoid ; the whole of the sella turcica may rest upon the sphenoidal sinus, and its anterior half may even lie over the most posterior ethmoidal cell. The sphenoidal sinuses and ethmoidal cells may border on both the foramen rotundum and foramen ovale. And as these sinuses may come into close relation with the structures that pass through the sphenoidal fissure, it seems a feasible explanation that disease of the bone walls of the sphenoidal sinuses and ethmoidal cells, as well as cavernous sinus thrombosis, may affect branches of the fifth nerve, as well as the motor nerves that go to the ocular muscles. Intracranial suppuration may also cause paresis of these motor nerves. Tumours are a frequent cause of oculo-motor paralysis. Excluding cases due to cavernous sinus thrombosis and intracranial suppuration, these nerve lesions are of comparatively rare occurrence in connection with accessory sinus suppuration. This comparative rarity is to be explained by the thickness of the sphenoidal walls, even when the cavity is exceptionally expanded.

The symptoms of asthenopia in ethmoidal suppuration have already been referred to. Moreau and Schröder have described accommodative asthenopia in cases of sphenoidal sinus suppuration. Lapersonne and Fisch have also reported cases in which intra-ocular tension was raised in consequence of accessory sinus suppuration.

Before passing on to consider briefly some points in the diagnosis of cases of visual disturbance produced through accessory sinus suppuration, it may be mentioned that traumatic lesions have occasionally been induced in the form of paraffin emboli. In treating saddle depressions of the nose by subcutaneous injection of warm liquid paraffin, and in the submucous injection of the same material in cases of ozæna, various authors have reported phlebitis, thrombosis, and emboli, and a condition of " paraffinome " in the eyelids. In a few cases thrombosis of the ophthalmic vein and embolism of the central artery of the retina have been recorded. These complications are avoided by using cold, sterile paraffin with a melting-point higher than the blood temperature, or even a fever temperature.

As regards the clinical diagnosis of a case of visual disturbance that may be associated with accessory sinus disease, two facts must be borne in mind. The fundus of an eye may appear normal, and yet a serious visual defect be present, or the field of vision be altered and scotomata be present. Again, the nasal cavities may appear normal

or but slightly altered in appearance while suppuration is nevertheless present in one or more sinuses. Also, notwithstanding good vision, hyperæmia and some swelling of the optic disc may be present.

And as accessory sinus suppuration in cases of visual disturbance may be an accidental coincidence, it is evident that only by careful investigation can a diagnosis be made, or even the symptoms present be brought to light. In some cases it is impossible clinically to diagnose as to the causal relationship between accessory sinus suppuration and visual disturbance. The kind of visual disturbance in such cases varies; the ophthalmoscopic picture, too, varies. At first, notwithstanding complete blindness, as in cases of fracture of the optic canal, there may be no change in the fundus. Optic atrophy only appears later.

In some cases of suppuration of the accessory sinuses, venous congestion, slight swelling, and blurred edges, are seen in the optic discs. The arteries, too, of the retina may be narrowed and the veins dilated. In a few cases a typical choked disc is present. True optic neuritis is not infrequent.

In a few cases unilateral and bilateral hemianopsia has been described; also a central, true or relative, scotoma.

In addition to the difficulty sometimes present, of discriminating whether in associated nasal disease and visual disturbance the nasal disease is the cause of an optic lesion, we have yet the other possibility to bear in mind—namely, that an optic lesion may be due to accessory sinus disease, and yet the accessory sinus disease produce no visible intranasal suppuration. Such latent empyemata can only be revealed by operative exploration. This we are justified in undertaking in those cases in which the ophthalmic surgeon finds symptoms pointing to a possible cause in the accessory sinuses. Thus there may be symptoms of a relative or absolute central scotoma, a varying degree of failing visual acuity, contraction of the visual field, or amaurosis. In such a case we may venture on exploring the posterior accessory sinuses, as cures have been reported in cases of this nature where latent suppuration was discovered. Disease of the sphenoidal sinuses is not uncommon; more rare is disease of the posterior ethmoidal cells alone; commoner than either is disease of both posterior ethmoidal cells and sphenoidal sinuses. In exploration, therefore, it is necessary to open up on the suspected side, intranasally, both the posterior ethmoidal cells and the sphenoidal sinus.

The association of optic disturbances with tumour growth in the

naso-pharynx is uncommon, but it does occur. Through communications of bloodvessels and of lymph channels the intracranial cavity may become infected.

A direct extension of a tumour on to the optic chiasma and motor nerves of the eye muscles has occurred in some cases. Not infrequently suppuration of the posterior accessory sinuses is found associated with a neoplasm. Orbital infections may occur secondarily to accessory sinus suppuration.

Birsch-Hirschfeld has laid emphasis on the early symptom of a central scotoma as characteristic of a tumour in the neighbourhood of the posterior accessory sinuses. This he ascribes to degeneration of the papillo-macular bundle of the optic nerve, caused by the toxic effects of venous congestion.

Berger, on the other hand, gives as characteristic symptoms of a tumour of the sphenoidal sinus, a narrowing of the visual field, first on the temporal side, then concentrically, and finally involving the macula.

Bull, also, on finding a tumour in the nose, and associated with it blindness on the temporal side of the field, with intact central vision, diagnoses the site of the tumour as in the sphenoidal sinus. Schmidt-Rimpler had a case in which both optic nerves were intact notwithstanding the infiltration of the whole body of the sphenoid with sarcoma. I have cited a case of mine on a par with this case. In Schmidt-Rimpler's case, however, the third, fourth, and sixth nerves were partially interrupted in their function.

Although intracranial tumours as well as intracranial suppurations are common causes of "choked disc," yet such intracranial complications may be present without causing a change in the appearance of the optic discs.

In conclusion I wish to point out some of the methods of treatment.

Although cures of optic neuritis and of oculo-motor paresis have been recorded in cases in which only conservative treatment was carried out, yet in most cases of disease of the accessory sinuses associated with visual disturbance a free opening up of the affected sinuses is indicated.

As the importance of various anatomical variations cannot be kept in mind too carefully, it may be well to give a résumé of such variations.

There are no fewer than nine procedures that may be required in opening up the anterior wall of the sphenoidal sinus. In this operation the opening up of the anterior wall of the sphenoidal sinus goes

hand in hand with the opening up of the posterior ethmoidal cells, except in cases where the ethmoidal cells are placed in series above the sphenoidal sinus. The anterior sphenoidal wall, for operative purposes, has two surfaces, a free nasal surface, and one covered by the ethmoidal cells. Hajek found a variation in the breadth of this entire wall of from 10 to 18 millimetres, the nasal part ranging between 2·5 and 7 millimetres, and the ethmoidal part between 6 and 10 millimetres. In measurements made by me, the extreme variation in the breadth of the whole wall ranged between 8 and 28 millimetres, the nasal part 2 to 14 millimetres, and the ethmoidal part 1 to 18 millimetres.

A direct operative procedure for opening up the anterior nasal wall of the sphenoidal sinus is possible under favourable circumstances. In other cases, the procedure to adopt when suspecting a latent suppuration is to remove the middle turbinate, lay free the anterior sphenoidal wall, and open up the posterior ethmoidal cells. When visual disturbances are caused by accessory sinus disease, the sphenoidal sinuses and ethmoidal cells may be equally responsible. Hence it is wise to open up both the posterior ethmoidal cells and the sphenoidal sinus, even if the operation be only exploratory for a suspected latent suppuration. And where suppuration is obviously present, treatment by cleansing with lotions without operating is often impossible. Even in those cases where the posterior ethmoidal cells lie above the sphenoidal sinuses, resection of the middle turbinate exposes the field of operation and enables the operator to "take his bearings."

In curetting or clearing out the contents of these cavities, particular caution must be exercised in working on the outer and upper walls, for the optic canal may have a wall in common with these cavities, or it may even run within their boundaries.

Both the sphenoidal sinuses and the posterior ethmoidal cells may be much expanded. The most posterior ethmoidal cell may extend over the middle line to the region of the optic nerve of the opposite side; passing by the optic canal, it may expand into the small wing of the sphenoid, and may even extend up to the orbital fissure and foramen rotundum. The sphenoidal sinus, too, may reach the optic nerve of the other side; anteriorly it may reach up to the maxillary antrum and ethmoidal bulla, and have a wall in common with them. It may also extend into the nasal septum for a distance of 15 millimetres anterior to the ostium of the sphenoid. Further, it may extend as a recess towards the pterygo-palatine fossa and into the clivus.

In endeavouring to reach growths, cysts, or accumulations of pus, situated intracranially, various routes have been used. It may be of interest to mention them.

In a boy of eleven years of age vision was reduced to one-sixth, and there was suppuration in the ethmoidal and sphenoidal sinuses ; Goris opened these up intranasally. As meningeal symptoms now appeared, he determined to open into the middle cranial fossa. By entering through the orbital cavity up to the optic foramen, and removing the posterior ethmoidal cells as well as the upper and lateral wall of the optic foramen, he was enabled to open up an accumulation of pus under the optic chiasma. A strip of gauze was inserted underneath the chiasma. The symptoms previously observed disappeared, and the visual power was uninjured. But on the fifth day the child died of meningitis.

Methods of reaching the region of the sella turcica are various. Luc, as the result of anatomical studies, favours the route through the maxillary and sphenoidal sinuses. Braun also favours the intranasal route, and shows that the sella turcica can be reached when the cavernous sinus has been raised up from its bed. Horzky reaches tumours of the hypophysis by trephining the lateral wall of the skull.

Schloffer, Eiselsberg, and Loewe, reach the same region by resecting the root of the nose, and so exposing the sphenoidal sinuses, through which they enter. Krause reached an imbedded bullet, which lay in the region of the optic chiasma, by trephining above the orbital cavity, and elevating the dura from the anterior cranial fossa. He recommends this route as favourable for removing cysts and tumours. Borchardt recommends the same route, only he proceeds intradurally. With reference to reaching the deep-lying recesses of the sphenoidal sinus, Loewe recommends the resection and turning forward of the root of the nose. Such operations are difficult and dangerous, because of the region and its important anatomical relations, and because of the risk both of escape of cerebro-spinal fluid and of meningitis.

THE END

BAILLIÈRE TINDALL AND COX, 8, HENRIETTA STREET COVENT GARDEN